Explaining the Variability of Antisaccade Performance in Healthy Participants

Thesis submitted for the degree of Doctor of Philosophy

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I hereby declare that this thesis has not been and will not be submitted in whole or in part to another University for the award of any other degree. This thesis is of my own composition, and the material contained within describes my own work.

Signature……

Alisdair Taylor

24th September, 2010.

In loving memory of my father, “Ken”

Table of contents

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Summary

In the antisaccade task participants are required to saccade to the mirror image location of a sudden onset target. As such, the task provides a powerful tool with which to investigate the cognitive processes underlying goal-directed behaviour. In healthy participants antisaccade errors (prosaccades directed towards the target) occur on approximately 20% of trials, and increased antisaccade error rate is widely used as a measure of "cognitive disinhibition" in clinical settings. One aspect of antisaccade performance that has received relatively little attention is the large variability in error rate typically observed within healthy participants. Whilst there are many studies describing increased antisaccade error rates in patient populations, there has been comparatively little research into what individual differences might underlie the dramatic variations that are observed within healthy participants. This thesis presents five papers, each of which explores potential sources of variability in antisaccade performance in healthy participants.

The first paper used a cueing manipulation to explore the extent to which individual differences in the ability to maintain the task goal in mind will influence antisaccade error rate. The second paper addressed the potential role of differences in motivation, by determining the extent to which antisaccade performance is moderated by a range of incentives. In paper 3, the role of strategic influences was investigated by altering the task instructions that participants were given. In paper 4, task instructions and working memory load were manipulated in order to determine their effect on antisaccade error awareness. The final paper, based on data gathered across the preceding experiments, explored the extent to which individual differences in factors such as working memory capacity, processing speed, and personality measures (schizotypy and impulsivity) correlated with antisaccade performance. Across all studies, the data is used to test predictions made by current parallel programming models of antisaccade performance. The data suggests that a range of "top-down" factors can influence antisaccade performance, but that the most important individual difference in explaining antisaccade error rate in healthy participants is prosaccade latency.

List of Figures

Literature Review

Overview

The ability to inhibit a habitual response to an external stimulus, and initiate instead an internally generated alternative response underlies our ability to carry out goal-directed, purposeful behaviour. The cognitive processes underlying such behaviour can be studied in the laboratory with the antisaccade task, in which participants are required to refrain from making a prosaccade towards a sudden onset target, and initiate a saccade in the opposite direction instead. Despite its widespread use, the cognitive processes underlying antisaccade performance remain unclear, and within healthy participants performance varies enormously across individuals. The broad aims of this thesis are to explore the cognitive processes underlying antisaccade performance in healthy participants and to determine the extent to which factors such as motivation and strategy use, and individual differences in personality, working memory and processing speed can account for variability in antisaccade performance within healthy adults.

Parts 1-5 of this literature review comprise the background and general introduction to the five articles that form the experimental work of the thesis. These sections describe in more detail the aims of the thesis, the theoretical context in which the research is based, and the empirical research on which it builds.

Part 1 concerns the role of eye movements in everyday behaviour and provides a rationale for using comparatively simple oculomotor paradigms to investigate the cognitive processes underlying complex purposeful behaviour. Part 2 provides a broad overview of the neural mechanisms underlying saccadic eye movements. The neural systems involved in saccade generation will be discussed briefly. Part 3 presents research concerning prosaccade performance and discusses its relevance to cognition and neural mechanisms of saccades. A model of saccade generation is also presented in this section. Part 4 reviews previous findings of antisaccade performance in non human primates and human patient and healthy populations. Models of antisaccade performance are described and evaluated. Part 5 presents the hypotheses of the thesis and introduces a series of five experimental articles that test these hypotheses.

Part 1 – The Role of Saccades in Everyday Behaviour

It has been estimated that during waking hours, we make 2-3 saccades per second, and therefore that over the course of a lifetime we will generate more saccades than heartbeats (Rayner, 1998). Most saccades are only a few degrees in size (e.g. Bahill et al., 1975), although much larger saccades do occur during certain activities (e.g. Land, Mennie, & Rusted, 1999). These saccades are interspersed with periods of fixation, during which vision is active. During saccades vision is effectively inhibited, as the high velocities reached by the eye during saccades would result in the visual image being "smeared" across the retina.

1.1. Why do we make saccades?

The concentration of rods and cones in the retina is non-uniform. By far the largest concentrations of cone cells (which are responsible for high acuity colour vision) are found on the comparatively small foveal area which corresponds to approximately 2-3 degrees of visual angle, whereas the entire visual field comprises approximately 200 degrees (Henson, 2003). By using saccadic eye movements, we are able to reorient the high acuity fovea to a different part of the visual scene, and thus quickly build up a more accurate internal representation of what we are viewing. In this way, complex scenes can be explored efficiently, as certain areas of particular relevance can be selected for more detailed processing whilst others can be ignored (Henderson & Hollingworth, 1999; Rayner & Pollatsek, 1992). Without a saccadic system, we would not be able to make accurate decisions regarding scene perception.

1.2. Why are saccades interesting to psychologists?

Carpenter (1994) argued that the oculomotor system provides researchers with a miniature model of the brain, one in which we can manipulate sensory input and accurately measure motor output with relative ease. Carpenter also argued that each saccade can be seen as reflecting the outcome of a decision process (Carpenter & Williams, 1995). Out of all the possible locations to which we could move our eyes, we have to select just one. The decision as to where to saccade next provides an important

insight into which aspects of the visual scene are most important or relevant, given a person's current goals.

Saccades towards sudden onset targets typically take around 150-200msec (Land & Tatler, 2009) but research in non-human primates suggests that it takes 60-80msec in total for a signal to be transmitted from the retina to the superior colliculus (SC) and then to the brain-stem neurons that in turn activate the oculomotor muscles initiating a saccade (Carpenter, 1981). According to Carpenter (1981, 2001) the extra 100-120msec reflects the decision making process referred to above. As will be explored in later sections this decision process is influenced by both "bottom-up" (external or exogenous factors) and "top-down" (internal or endogenous factors).

Part 2 – Neuroanatomy of Saccades

Evidence from neurophysiological studies in non-human primates, the behaviour of patients with neurological disorders or lesions to specific brain areas, and functional neuroimaging studies in healthy participants have led to a sophisticated understanding of the neural mechanisms underlying saccadic eye movements.

The saccadic system comprises a complex network of cortical and sub-cortical brain regions. In brief, signals from the rods and cones in the retina are passed down the optic nerve to the visual cortex via the lateral geniculate nucleus. Different regions of the visual cortex are responsible for basic visual processes such as edge detection. The visual cortex projects to the temporal cortex via the ventral stream, and to the parietal eye fields, via the dorsal stream. In turn, these areas project to frontal areas including the frontal eye fields, supplementary eye fields, and the dorsolateral prefrontal cortex (e.g. Pierrot-Deseilligny, Ploner, Müri, Gaymard, & Rivaud-Pechoux, 2002). These frontal and parietal regions themselves project to several thalamic and basal ganglia areas, including the caudate nucleus, substantia nigra, thalamus and sub-thalamic nuclei (Scudder, Kaneko, & Fuchs, 2002). In turn, these areas project to the superior colliculus, which itself projects to the saccadic nuclei that together form the "brain stem saccade generator" and which provide the final common pathway innervating the extraocular muscles to effect saccadic eye movements (see figure A). The following sections will consider each aspect of this pathway in more detail.

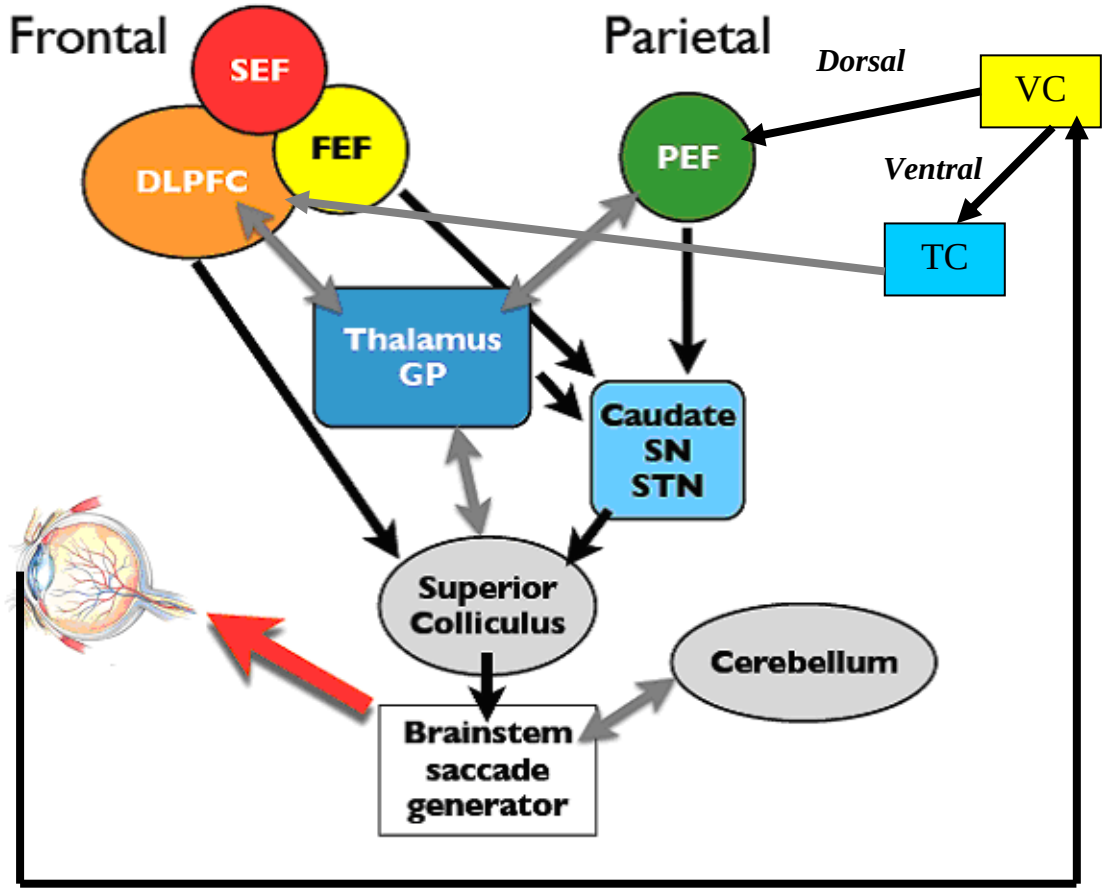

Figure A. The complex neural structure of saccade generation

Note: the retina receives visual information that is transferred to the visual cortex and eventually the brainstem saccade generator sends saccade information to the oculomotor muscles of the eye, and the saccade is made.

VC = Visual cortex, TC = Temporal cortex, PEF = Parietal eye field, SEF = Supplementary eye field, FEF = Frontal eye field, DLPFC = Dorsolateral prefrontal cortex, SN = Subthalamic nuclei, STN = Substantia nigra, GP = Globus pallidus.

2.1. Oculomotor nuclei in the brain stem

As mentioned above, saccadic eye movements are controlled by a complex network of cortical and sub-cortical structures. Saccades are elicited by activation in burst neurons (BNs) within the 'brain stem generator' (see figure A). BNs show an intense discharge before each saccade and project monosynaptically to ocular motor neurons in the abducens nucleus (Scudder et al., 2002). BNs in the rostral interstitial

nucleus of the medial longitudinal fasciculus (riMLF) discharge before vertical saccades. For horizontal saccades, BNs discharge in the paramedian pontine reticular formation (PPRF). Omnipause neurons (OPNs) tonically inhibit BNs and the former are located in the nucleus raphe interpositus around the midline of the caudal pontine reticular formation (Büttner-Ennever, Cohen, Pause, & Fries, 1988). OPNs pause during and before saccades in all directions and discharge at a high tonic frequency between saccadic eye movements (Raybourn & Keller, 1977). The activity of OPNs reflects the decision to either generate a saccade or to maintain fixation (Everling & Fischer, 1998).

2.2. Superior Colliculus

The superior colliculus (SC) is a critically important area for saccade generation. Microstimulation studies have revealed that it contains a saccadic 'motor map' in its intermediate layers and a topographic 'retinal map' in its superficial layers (Everling & Fischer, 1998). The saccadic motor map contains cells which determine the direction and size of a saccade. Importantly, neurons in the rostrolateral pole in the intermediate layers of the SC pause before and during saccades and are tonically active during fixation (Munoz & Wurtz, 1992). In other words, the SC contains both fixation and saccade related neurons, (Everling, Pare, Dorris, & Munoz, 1998). Saccade related neurons in the SC are tonically inhibited by GABAergic neurons in the substantia nigra pars reticulata (SNpr). For saccadic eye movements, neurons are activated in a region that represents the point to which the saccade will be directed. This description can be likened to a peak in a hill, where by neural activity in the SC uses a small fraction of the topographical map to form a hill and the peak in the hill corresponds to the saccade target. Similarly, cortical areas which project to the SC, such as the frontal eye fields (FEF), contain motor maps, whereby the locus of stimulation will determine the direction and amplitude of the subsequent saccade. Fixation related neuron activity has also been observed in the FEF (e.g. Bruce & Goldberg, 1985). In sum, the main goal of the SC is to translate visual information from the retina into oculomotor information, thus eliciting a saccade to the target or a location of interest.

2.3. Cortical regions

2.3.1. Posterior parietal cortex (PPC)

As mentioned earlier, many cortical areas can influence saccade generation. The parietal cortex has been found to be involved with saccade generation for a variety of saccadic paradigms (see McDowell, Dyckman, Austin, & Clementz 2008 for review). Specifically, evidence suggests that the parietal eye fields (PEF) (an area within the posterior parietal cortex), is involved in the preparation and initiation of saccadic eye movements (Barash, Bracewell, Fogassi, Gnadt, & Andersen 1991). For example, patients who have lesions to the PEF have increased saccade latencies compared to healthy controls (Pierrot-Deseilligny, Rivaud, Gaymard, & Agid, 1991a). Previous research has found evidence to suggest that the posterior parietal cortex (PPC) is particularly involved with establishing the spatial co-ordinates of saccades (figure B), (see Pierrot-Deseilligny, Rivaud, Gaymard, Müri, & Vermersch, 1995 for review). For example, Pierrot-Deseilligny, Rivaud, Gaymard, & Agid (1991b) found increased error rates in patients with lesions to the PPC on a memory-guided saccade task. This task requires participants to store and calculate the spatial co-ordinates of the intended saccade in working memory (see section 3.6.2). The parietal eye fields (PEF) play a key role in saccade generation too. As the parietal eye fields project directly to the superior colliculus, it has been argued that "express saccades" (prosaccades to sudden onset targets with very short latencies between 80 and 120msec) may be initiated via this route (Fischer, 1986; Fischer & Ramsberger, 1984).

2.3.2. Frontal eye field (FEF)

Another cortical area of particular importance to saccade generation is the frontal eye field (FEF). As with the PEF the FEF projects directly to the brainstem saccade-generating circuitry (e.g. Seagraves, 1992). Numerous functional neuroimaging studies have found increased activity in the FEF during saccades (see McDowell et al., 2008 for review). For example, McDowell et al. (2005) investigated cortical activity preceding prosaccades and antisaccades using combined electroencephalography (EEG) and magnetoencephalography (MEG). They found that presaccadic activity (activity prior to saccade generation) in the medial FEF was greater for antisaccades compared to prosaccades. Support for the involvement of the FEF in saccade generation can be found

in non-human primate research (e.g. Everling & Munoz, 2000; Hanes & Schall, 1996). Using single neuron recording techniques, these studies found a relationship between pre-target activity in the FEF and the time it takes the SC to discharge saccade-related neurons. The lower the pre-target activity in the FEF, the longer it takes to boost the firing rate in SC neurons past the threshold for triggering a saccade. It is possible that one factor explaining variability in saccade latencies could be different levels of FEF activity.

2.3.3. Supplementary eye field (SEF)

Another important frontal area for saccade generation is the supplementary eye field (SEF), situated anterior to the FEF (Leigh & Zee, 1999). The SEF has connections to cortical areas required for simple eye movements, such as a standard prosaccade. These areas include parietal areas, such as the parietal eye field (PEF) and the superior parietal lobe. Furthermore, the SEF has reciprocal connections to the frontal eye fields (FEF). In humans, previous research has found that microstimulation of the SEF results in saccade generation (Godoy, Luders, Dinner, Morris, & Wyllie, 1990). Converging evidence from single neuron recordings in monkeys and neuroimaging studies in humans have found saccade-related activity in this area (e.g. Brown, Goltz, Vilis, Ford, & Everling, 2006; Dyckman, Camchong, Clementz, & McDowell, 2007). Previous research has found that activity in the SEF arises during saccade generation in prosaccade tasks (e.g. Luna et al., 1998; McDowell et al., 2005). Interestingly, activity in this region is normally greater when complex saccade paradigms are used (e.g. antisaccades) (e.g. Luna et al., 2001; Raemakers, Vink, Van den Heuvel, Kahn, & Ramsey, 2006; Reuter, Kaufmann, Bender, Pinkpank, & Kathmann, 2010). In addition, the SEF is vital for saccadic tasks that require a motor sequence or when the stimuli are predictable such as in a sequential memory-guided saccade task (Gaymard, Rivaud, & Pierrot-Deseilligny, 1993). Finally, Munoz & Everling (2004) suggest that the SEF plays a key role in the preparation of an antisaccade. They argue that during antisaccade generation, activity in movement neurons in the SEF is increased, which helps facilitate an antisaccade.

2.3.4. Dorsolateral prefrontal cortex (DLPFC)

Finally, there is some evidence to suggest that the dorsolateral prefrontal cortex (DLPFC) may also have some relevance to saccade generation (see Pierrot-Deseilligny,

Müri, Ploner, Gaymard, & Rivaud-Péchoux, 2003 for review). Several functional neuroimaging studies have suggested its involvement is greater with more complex saccade paradigms, such as with memory-guided saccades, compared to simple prosaccade tasks (e.g. Sweeney et al., 1996). Using transcranial magnetic stimulation (TMS), Müri, Vermersch, Rivaud, Gaymard, & Pierrot-Deseilligny (1996) found that stimulating the DLPFC during the spatial memory stage of a memory-guided saccade task (see section 3.6.2.), increased the spatial error of subsequent memory-guided saccades. Support for the lack of involvement the DLPFC has with simple prosaccade paradigms is taken from lesion studies. These studies have found no change in prosaccade performance for patients with lesions to the DLPFC compared to healthy controls (Pierrot-Deseilligny et al., 1991b; Pierrot-Deseilligny et al., 2003).

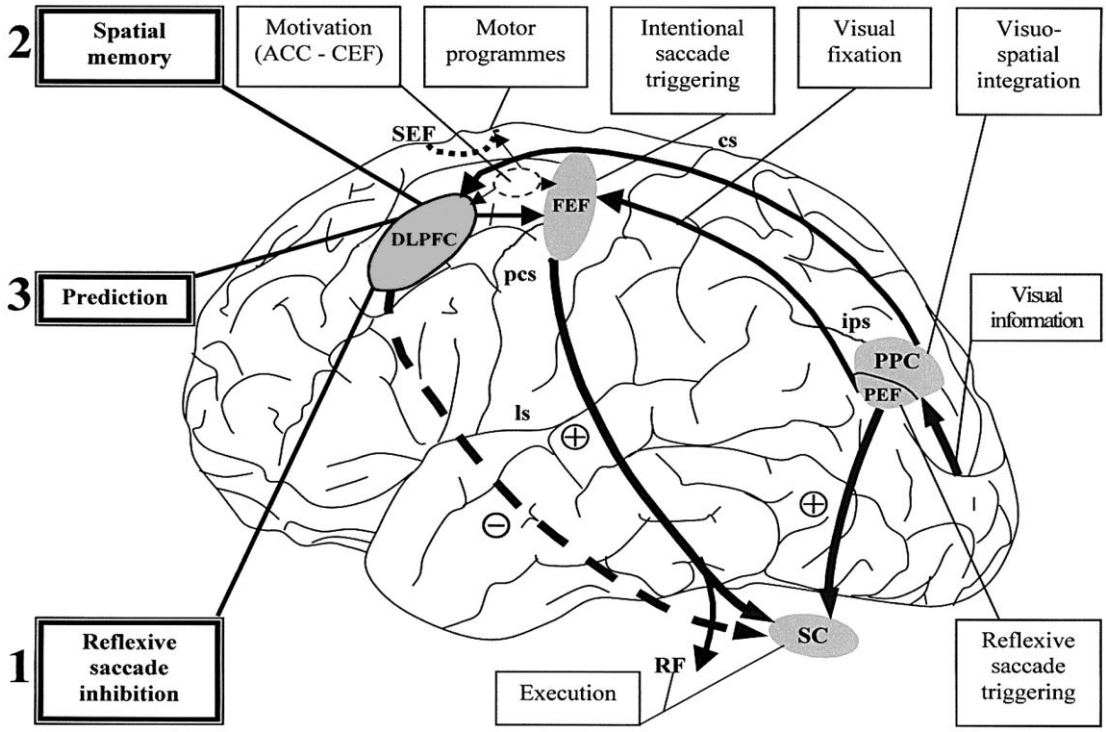

Figure B. Characteristics of the cortical areas involved with saccades
Taken from Pierrot-Deseilligny et al. (2003b).

Having briefly outlined the neural mechanisms underlying saccade generation in this section, the following section considers the relationship between cognitive processes and visually guided saccades.

Part 3 – Prosaccades & Cognition

The previous sections have referred to saccadic eye movements in general. In the laboratory researchers have used a range of different saccadic tasks to explore the relationship between saccades and cognitive processes. The "prosaccade task" is one of the simplest, and is used to describe a task in which participants make a visually guided saccade towards a sudden onset target. In the next sections studies that have explored the effects of exogenous and endogenous factors on prosaccade performance will be discussed and the extent to which these findings can be explained by models of saccade generation will be addressed.

3.1. Prosaccades

In a typical prosaccade task, participants' are required to focus their gaze on a central fixation point and then quickly and accurately saccade towards a suddenly appearing target stimulus. Typically, the time it takes to make a prosaccade (correct latency) is normally around 190msec and healthy participants make few errors (saccading away from the target) (Hutton, 2008). The latency of prosaccades can vary to a great degree on a trial by trial basis (from about 120msec to 220msec) depending on a wide range of factors, Mosimann, Felblinger, Colloby, & Müri, 2004). Interestingly, patient populations do not normally exhibit deficits on prosaccade performance compared to healthy controls (see Hutton & Ettinger, 2006), although as mentioned earlier, lesions in the parietal eye field (PEF) can lead to reduced accuracy of prosaccades (Pierrot-Deseilligny et al., 1991a).

3.2. LATER model

Carpenter (1981) then later Carpenter & Williams (1995) developed an important model for interpreting saccade generation. Their Linear Approach to Threshold with Ergodic Rate model, or LATER for short, provides a useful framework within which to consider the influence of cognitive processes on saccade generation. Carpenter's model applies an accumulator model of decision making to saccade generation. Like other decision models, the LATER model assumes two things. Firstly,

an accumulation of information concerning the different potential responses occurs over time and secondly, a decision is reached when information concerning one possible response reaches a critical threshold before the others (or the decision not to respond is made if no threshold is reached).

The LATER model proposes three key parameters, a baseline level of activity, a threshold level of activity above which a saccade is generated and the "rate of rise" of the decision signal. According to the model, the decision signal rises linearly at a rate (r), starting from the baseline level of activation (S_0) and finishing at the threshold for triggering a saccade (S_T). It is assumed that the decision signal starts to rise at target onset. The rate of rise in activity is assumed to vary randomly from trial to trial, and according to Carpenter, this variation is what explains the considerable variation in prosaccade latency within healthy participants.

According to the LATER model, a manipulation that affects the threshold for triggering a saccade will impact on the distribution of the saccade latencies (Reddi & Carpenter, 2000). Similarly, a manipulation that results in changes in the baseline level of activity, (i.e. expectations, see Oswal, Ogden, & Carpenter, 2007), or the rate of rise of activity could also result in changes in saccade latency or saccade distribution. For instance, in a standard saccade task, Oswal et al. (2007) found that increasing the foreperiod (the interval between warning signal and stimulus) resulted in significant differences in the latency distributions, whereas decreasing the foreperiod did not. As the foreperiod increased, median latencies decreased progressively. The authors concluded that these changes in distribution and latencies for increased foreperiod trials may have arisen from increased expectation or prior probability. Therefore, in terms of the LATER model, latencies may be reduced as a result of expectancy increasing the initial level of baseline activation, which in turn means that the rise in activity from baseline level to the threshold for saccade triggering has a shorter distance to go before reaching the threshold for triggering a saccade.

Neurophysiological studies have also provided support for the LATER model. For example, Dorris, Pare, & Munoz (1997) found a correlation between the rate of increase in neural activity in neurons in the motor region of the superior colliculus (SC) and saccade latencies in non-human primates. Their finding suggests saccade initiation is likely to occur when activity is high enough, supporting the accumulator concept of the LATER model. Ultimately, the LATER model is a good starting point for attempting to explain saccade generation and provides insight into the decision

processes involved when making a saccade. Furthermore, the LATER model has provided the basis for some current models of saccade performance (e.g. competitive race models (see section 4.4.2.).

3.3. Top-down vs. Bottom-up processes in saccade generation

One of the advantages of the LATER model is that it allows for both exogenous (bottom up) and endogenous (top down) factors to influence saccade latency. Bottom-up factors represent information such as the luminance, size and position of a target stimulus whereas top-down processes reflect factors such as the goals or expectations that the observer has.

The remainder of this section summarises a number of key manipulations that have been found to impact on prosaccade performance. These manipulations have been divided into either 'bottom-up effects' because they primarily reflect exogenous factors or 'top-down effects' because they are more associated with endogenous processes.

3.4. Bottom-up effects on prosaccade performance

3.4.1. Gap effect

One manipulation that has been used to explore bottom-up effects on prosaccade performance is to vary the amount of time between the offset of the central fixation point stimulus and the onset of the target stimulus. In a standard 'step' paradigm, the offset of the fixation point coincides with the onset of the target. In an 'overlap' paradigm, however, the central fixation stimulus remains visible for a brief period after target onset. Finally, in the 'gap' paradigm, the fixation offset is followed by a gap (normally of 200msec) which precedes the target onset. The critical finding is that, compared to step trials, correct prosaccade latencies are often increased in overlap trials and decreased in gap trials (e.g. Fischer & Weber, 1992; Reuter-Lorenz, Hughes, & Fendrich, 1991). Fischer & Weber (1993) argue that latencies are faster in the gap paradigm because the disappearance of the central fixation stimulus acts as a 'quick release' to disengage attention away from this location thus allowing it to be allocated more rapidly to the target when it subsequently appears. In an overlap paradigm,

latencies should be slower, as attention remains “glued” to the fixation point at the time of target onset.

Support for the role of attention when explaining the decrease in correct latencies on gap trials, or ‘the gap effect’ as it is called, comes from a recent study by Pratt, Lajonchere, & Abrams, (2006). In their study, participants had to attend to a portion of a complex fixation stimulus by focusing attention to a specified line segment. The fixation stimulus comprised two line segments that made a cross when put together and the intersection of the crossing lines remained blank. One line was green and one was purple. Participants were instructed to fixate at the centre of the cross. 200msec after fixation, one of the lines disappeared, then reappeared, then disappeared, then finally reappeared. Each disappearance and reappearance lasted for 100msec. This was done in order to draw attention to the specified line segment. Following this, participants were asked to maintain fixation for a further 1000msec and then a warning tone was presented for 100msec. The fixed tone interval alerted participants to the appearance of a target. The offset of the warning signal was when ‘saccade trial’ or ‘key press trial’ would appear on screen. For saccade trials, there were five possibilities. Either no gap, overlap (the cross stimulus overlapped with the presentation of the onset target), full gap (cross disappears), gap attended (designated line remains during gap interval) or gap unattended (non designated line remains during gap interval). Gaps were 200msec. On key press trials, no eye movement was required. After a 200msec gap, one of the lines (designated or non-designated) that comprised the cross stimulus either shrunk or grew. Participants pressed ‘l’ on the keyboard if they thought it had grown and pressed ‘z’ if they thought it had shrunk. Saccade trials were used to measure the gap effect and key press trials were used to confirm that participants complied with the attention instructions. Pratt et al. (2006) found that when the unattended portion of the central stimulus was removed (i.e. the non-specified line segment) 200msec prior to the target onset, participants showed a smaller gap effect, compared to when the attended portion was removed. The authors interpret this finding in terms of fixation neuron activity. They suggest that within the superior colliculus (SC), fixation neuron activity is modulated by attentional selection. Specifically removing the unattended portion of the central cross (as opposed to removing the attended portion) will elicit greater inhibition of collicular movement cells, causing a smaller reduction in the latency of saccades to the onset target. However, if the attended portion of the stimulus is removed prior to target onset, then fixation activity is

decreased, movement cells are disinhibited and saccades to the target are initiated much faster, hence the ‘gap effect’.

Some researchers have suggested that the gap effect represents both a ‘warning’ and ‘fixation release component’ (Kingstone & Klein, 1993; Reuter-Lorenz, Oonk, Barnes, & Hughes, 1995). These authors argue that the offset of the central fixation stimulus acts as a cue that warns the participant that the target is about to appear, resulting in a faster initiation of the saccade to the target. According to Reuter-Lorenz et al. (1991), fixation neurons in the Superior colliculus (SC) will become less active when the fixation stimulus is extinguished, which enables movement neurons to facilitate the initiation of the next saccade.

A recent study by Vernet, Yang, Gruselle, Trams, & Kapoula (2009), looked at prosaccade performance in young (21-29) and middle (39-55) aged adults. Consistent with the gap effect, they found reduced latencies in gap trials compared to overlap trials for both age groups. Interestingly, when the middle aged group performed gap and overlap prosaccades that were interleaved in the same block, correct prosaccade latencies were increased in all directions (left, right, up, down) compared to when gap or overlap trials were performed alone. However, performing mixed gap and overlap prosaccade trials increased latencies for the young adult group when the direction of the saccade was right and decreased latencies for all other directions (left, up, down). The authors concluded that the reason for this difference in mixed gap and overlap prosaccade performance is because of differences in frontal eye field (FEF) activity between the two age groups. They argue that frequent changes in activity (because of switching between gap and overlap trials) may be more time consuming for a ‘less optimally functioning’ FEF, as in the case of middle aged adults. Whereas frequent changes in FEF activity may not be so detrimental and even capable of reducing latencies for an FEF that is functioning optimally, as in the case of young adults. Research into the alternation of gap and overlap tasks provides a way of exploring bottom-up effects on prosaccade performance.

In sum, gap tasks reduce the time it takes to saccade to the impending target compared to step or overlap tasks, but the extent to which attention plays a significant role in this effect remains a topic of debate.

3.4.2. Exogenous cueing

Another exogenous factor that has been shown to modulate prosaccade performance is cueing the goal location of the 'to be made' saccade. It should be noted that a cue can be either exogenous or endogenous. Centrally positioned cues, whose meaning requires interpretation, (such as an arrow), are generally considered to be endogenous, and cues of this type are addressed in the next section. For the purposes of this section, research that has looked at the effects of exogenous cues (also called peripheral cues) on prosaccade performance will be considered. An exogenous cue is normally a cue situated nearer to the area that is being cued, for example, a momentary flash of the box that surrounds the target stimulus.

Several studies have found that cueing the correct location (i.e. the location at which the target stimulus appears), results in reduced prosaccade latencies (e.g. Cavegn, 1996; Fischer & Weber, 1998). A straightforward interpretation of this finding is that the cue serves to shift attention to the target location, resulting in faster target detection when it subsequently appears (Müller, 1994; Posner, 1980; Remington, 1980). Conversely, cueing the incorrect location (i.e. the location opposite to where the target stimulus appears), has been found to increase correct prosaccade latencies (Walker, Kentridge, & Findlay, 1995). In this case, it is assumed that attention is allocated to the cued location, and the necessity of disengaging attention from the cued location and shifting it towards the target location incurs an extra time cost. Taken together, these results further support the close relationship between saccadic programming and spatial attention suggested by the gap effect.

Some studies have investigated the effects of cue lead time (CLT) on prosaccade performance. CLT is the amount of time between the offset of the cue and the onset of the target stimulus. In a study by Fischer & Weber (1998) participants performed prosaccades where a cue (a momentary flash of the flanker box that surrounded the target stimulus or the location opposite to the stimulus) was briefly presented before the appearance of the target stimulus. In separate blocks, cues were either presented to the opposite side of the target stimulus (anti-cue), or presented at the same side as the target (pro-cue). In addition, various CLT's were used (100-700msec). In the anti-cue block, they found that as CLT increased, more anticipatory saccades (saccades which are < 80msec, Wenban-Smith & Findlay, 1991) were made and correct prosaccade latencies increased. In the pro-cue block, they found that latencies were decreased when 100msec CLT was used compared to no CLT. This 'cueing effect' is consistent with studies in

the attentional literature (Posner & Cohen, 1984), who have also found cueing effects on reaction times using short CLT's. The increase in correct latencies for trials with long CLT's, found by Fischer & Weber (1998), have been referred to as 'inhibition of return' (Klein, 2000). Inhibition of return (IOR) is reduced attentional priority for information in a region that recently experienced a higher priority of attention; therefore IOR biases attentional orienting away from previously inspected locations. Different interpretations of IOR have been given. For instance, Klein (2000) has suggested that IOR is a foraging facilitator, used to facilitate effective visual search, by preventing us returning to recently inspected locations using an inhibitory mechanism. However, Hooge, Over, van Wezel, & Frens (2005) have found evidence to suggest that IOR is not used as a foraging facilitator, as they found increased fixation latencies to locations that had been previously attended to.

3.4.3. Distractor stimuli

Some studies have explored the impact of task-irrelevant distractors on prosaccade performance. For example, Theeuwes, Kramer, Hahn, & Irwin (1998), asked participants to make prosaccades to one of six target stimuli, arranged in a circle around a central fixation stimulus, in order to discriminate a small target contained within the circles. On all trials, all but one of the six peripheral circles changed from grey to red. At the same time an additional red distractor stimulus appeared at one of four additional locations on half of the trials. A prosaccade had to be made to the colour singleton (the remaining grey circle). Detection time at the colour singleton was significantly increased, in trials containing a distractor stimulus, and eye tracking revealed that this occurred because participants often made a saccade near or on the distractor location, followed rapidly by an additional saccade that took the eye to the singleton. The authors also found that pre-cueing the location of the colour singleton eliminated saccades to the distractor location. They argued that the precue allowed a saccade to be facilitated towards the location of the colour singleton (as opposed to the location of the distractor) as they believed the pre-cue shifted attention to the location of the colour singleton in advance of the distractor's appearance.

Distractor stimuli have been shown to affect other prosaccade metrics. For example, Doyle & Walker (2001) asked participants' to make vertical prosaccades towards a target stimulus presented either above or below the central fixation point. On some trials, a distractor stimulus was presented on either the left or right of the central

point. They found that regardless of target location, the presentation of a distractor stimulus on either the left or right resulted in marked curvature of the saccade path away from the side on which the distractor was presented. These results suggest that even when the saccades themselves are spatially accurate, distractor stimuli can result in deviations in saccade trajectories. McSorley, Haggard, & Walker (2006) found that the direction of the saccade curvature largely depends on preparatory processes that occur before the onset of the distractor. Essentially, if there is insufficient time to prepare a saccade, the activity in the oculomotor system caused by the distractor is not inhibited, and instead competes with the activity caused by the target, causing curvature towards the distractor. Alternatively, if sufficient time is allowed for a saccade to be prepared to the target location, the distractor location is inhibited, resulting in curvature away from its location.

Interestingly, when a target stimulus and distractor stimulus are presented at the same time, and reasonably close together, saccades are often made to a location in between the two (Findlay, 1982). This "centre of gravity" effect can be interpreted in terms of salience maps. As mentioned earlier in this review, the superior colliculus contains a topographic retinal map (visual hemisphere) and a saccadic motor map for determining the direction and size of a saccade. Normally when a target is presented alone, neural activity in the SC uses a small fraction of the topographical map to form a hill and the peak in the hill corresponds to the saccade target. However, because a distractor is presented as well, neurons are activated in the region containing the distractor, causing an additional peak. Presumably, in these situations, the co-ordinates of the average distance between the target and the distractor are used, thus facilitating the majority of saccades to this 'in between' target and distractor location.

3.4.4. Stimulus eccentricities

Varying stimulus eccentricities (i.e. target location in degrees) has also been shown to impact on prosaccade performance (e.g. Kalesnykas & Hallett, 1994; Weber, Aiple, Fischer, & Latanov, 1992). Several studies have observed that saccade latencies are faster to targets which are closer to the central point, compared to targets which are further away from the centre (e.g. Biguer, Prablanc, & Jeannerod, 1984; Hallett & Lightstone, 1976; White, Eason, & Bartlett, 1962). However, a somewhat different pattern of results was found in a recent study using non-human primates.

Bell, Everling, & Munoz (2000) explored the effects of different stimulus eccentricities on prosaccade performance in two non-human primates. The authors used 5 eccentricities (2, 4, 8, 10 & 16° from the central point). They found that saccade latencies decreased as target eccentricities were increased from 2 to 10°. However, when eccentricities were increased further, i.e. to 16°, latencies increased. The authors suggest that the increase in latencies for eccentricities with larger amplitudes (i.e. > 10°) could be due to reduced visual acuity. They argue that increasing the eccentricity results in its location on the retina moving away from the fovea, resulting in reduced visual acuity, resulting in longer latencies for saccades with large amplitudes. In addition, saccades to eccentricities below 8° may experience long latencies because of the additional time needed to disambiguate fixation and saccade signals contained in the superior colliculus (Munoz & Wurtz, 1995). The results suggest that in humans at least, and in terms of the LATER model, the rate of rise of the decision signal of the prosaccade is speeded up and will reach the threshold for triggering a saccade much faster if smaller eccentricities of targets are used (e.g. 2-14°) compared to larger eccentricities (e.g. prolonged latencies for eccentricities which are > 15°, Kalesnykas & Hallett, 1994).

3.4.5. Saccade direction

Another bottom-up factor that has been shown to influence prosaccade performance is saccade direction (e.g. Bell et al., 2000; Goldring & Fischer 1997). Dafoe, Armstrong, & Munoz (2007) gave participants prosaccades where participants had to either saccade to a suddenly appearing target on the horizontal axis, or the vertical axis. The authors found that correct prosaccade latencies were faster for saccades made horizontally compared to vertically. In addition, prosaccades to the upper hemifield were faster than to the lower hemifield. Bell et al. (2000) offer an evolutionary explanation for the above mentioned upper hemifield bias. They suggest that the oculomotor system may have adapted to favour faster orienting movements towards certain locations over others, in order to maximize efficiency for specific behaviours, e.g. non-human primates scanning for predators. Dafoe et al. suggest that studies investigating prosaccade performance should consider saccade direction, i.e. not just focus on horizontal saccades.

The research described above has highlighted a number of exogenous factors that have been shown to alter performance (mainly latencies) on simple prosaccade

tasks. The next section outlines a number of endogenous or ‘top-down’ factors that have also been found to influence prosaccade generation.

3.5. Top-down effects on prosaccade performance

A number of studies have shown that prosaccade performance can be affected by endogenous as well as exogenous factors. These “top-down” influences are reviewed in the following sections.

3.5.1. Probability

By varying the probability with which a target appears at a specific location, we are able to understand how learning may mediate prosaccade performance. Carpenter & Williams (1995) altered the probability of a target stimulus appearing on either the right or left of the central fixation point. They found that correct prosaccade latencies were faster to the left target than the right target, when the probability of the target appearing to the left of fixation was higher than the probability of the target appearing to the right of fixation. The same result was found when target probability was higher to the right of fixation. The authors suggest that if participants’ expectation of the location of the intended saccade is increased, then this will reduce saccade latencies because the increased expectancy raises the baseline level of activation, resulting in a shorter distance for the rate of rise in activity to reach the threshold for triggering the saccade. Therefore, if expectation is higher for the left side (because the probability of the target appearing there was higher than on the right), then the baseline level of activation will begin at a point which is closer to the threshold for triggering the saccade on left sided trials compared to trials on the right side.

In a more recent study, Dick, Kathmann, Ostendoorf, & Ploner (2005) explored the effects of target probability on prosaccade performance. In a within-participants design, participants performed a gap prosaccade task (central fixation point extinguished 200msec before target onset) and a ‘warning prosaccade task’ (central fixation point changes colour 200msec before target onset, but remains illuminated until target appears). The experimenters varied the probability of which the target stimulus would appear. In low probability blocks, saccade targets appeared in 25% of all trials, making 75% of trials catch trials and vice versa for high probability blocks. On catch trials,

participants had to keep fixation until the beginning of the next trial, without making a saccade. Correct prosaccade latencies were faster in blocks that contained a higher probability of the target appearing (75%) compared to blocks which contained a lower probability of the target appearing (25%). This effect was found in both the gap task and the warning task. In addition, the distributions of latencies were different between gap trials and warning trials. On gap trials, discrete changes of saccade latencies were found. In the warning task a shift of the entire latency distribution towards longer latencies with low target probability was found.

The authors explain the difference in probability-dependent changes in latency distributions between tasks in light of Carpenter & Williams (1995) LATER model of saccade generation. As previously highlighted, Carpenter & Williams suggest that a saccade is made when a decision signal reaches the desired threshold for triggering the saccade. The LATER model predicts that manipulations capable of influencing the level of the threshold, or the rate of rise in activity of the decision signal, or the level of baseline activation, will impact on the variability of prosaccade latencies. In line with this model, Dick et al. (2005) suggested that a change in the rate of rise of activity of the decision signal could account for the distribution of latencies in the warning task and a change to the threshold could account for the latency distribution in the gap task. The authors assume that changes in fixation activation are the reason for this difference in explanations for the distributions in the gap and warning tasks.

Other studies investigating the effects of probability on prosaccade performance have mixed prosaccade trials with other types of task. For example, in their first study, Olk & Kingstone (2003) gave participants a block of prosaccades and a separate block of prosaccades mixed with antisaccades (see section 4.4.2). They found that correct prosaccade latencies were faster when prosaccades were completed alone, compared to when they were completed in a block with antisaccades. Similarly, several studies have reported an increase in prosaccade errors when prosaccades are performed with other saccadic tasks (e.g. Barton, Raoof, Jameel, & Manoach, 2006; Cherkasova, Manoach, Intriligator, & Barton, 2002; Reuter, Philipp, Koch, & Kathmann, 2006). One possible explanation for these findings, is that performing prosaccades in a mixed design with another task, increases the demands of attentional resources for making prosaccades. When performing prosaccades in a mixed block, the upcoming trial is not predicted by the previous trial, which ultimately places a cost on the active maintenance of the task goal (Unsworth, Schrock, & Engle 2004).

3.5.2. Endogenous cueing

Butler & Zacks (2006) looked at the effects of cueing on prosaccade performance in a sample of young and older adults. The authors varied the type of response cue, (peripheral onset, vs. central arrow). In the peripheral onset condition (exogenous), participants were asked to make a prosaccade to a target stimulus that appeared in one of two flanker boxes to the left and right of the fixation cross and the exogenous cue was represented by one of the flanker boxes turning white. In the central arrow condition (endogenous), again, participants were asked to make a prosaccade to a target stimulus that appeared in one of two flanker boxes and the endogenous cue was represented by an arrow pointing towards one of the two flanker boxes. The authors found no effect of cue type (peripheral vs. central cues) on prosaccade accuracy or prosaccade latency in young adults and older adults. Peripheral and central arrow cues had an equivalent effect on prosaccade accuracy and latencies in both age groups suggesting that the extraction of additional visual information (cue) had no direct influence on saccade generation. However, in both age groups, prosaccade latencies were reduced (all be it not significantly) for peripherally cued trials compared to trials containing a central arrow cue. This finding suggests that the endogenous processing involved with central arrow cues, adds slightly more time to the decision process to make a prosaccade. One criticism of this study is that the authors did not include catch trials or uncued trials. Uncued trials could make it more difficult for participants to predict the direction of the intended saccade. Other researchers have interpreted the increase in latencies for endogenous cues compared to exogenous cues as reflecting the additional processing requirements of establishing the appropriate stimulus response mapping given the symbolic cue (Walker, Walker, Husain, & Kennard 2000).

3.5.3. Task instructions

Mosimann, et al. (2004) investigated the effects of varying task instructions on prosaccade performance. They found an increase in correct prosaccade latencies and errors in terms of gain when participants were asked to delay making a prosaccade (delay condition) to an impending target compared to when they were given standard instructions (make a prosaccade as quickly and as accurately as possible). In addition, the authors found that instructing participants to be spatially inaccurate (inaccuracy condition) resulted in slower correct latencies compared to when given standard instructions. These findings suggest that participants were using top-down mechanisms

to volitionally delay making a saccade (delay condition) and to perform poorly (inaccuracy condition), and not just bottom-up information, such as stimulus properties. These findings confirm the suggestion of Carpenter that saccades should not be considered truly "reflexive" as they reflect a complex interplay of bottom-up and top-down influences.

In sum, the research presented above clearly highlights a relationship between prosaccades and cognition. Specifically, a growing body of research has found evidence to suggest that a range of bottom-up and top-down factors can influence prosaccade performance, as outlined above. These findings emphasise the link between prosaccades and attention but the precise nature of the relationship remains unclear.

3.6. Volitional prosaccade tasks

Although it is clear that top-down factors play a role in prosaccade performance, a variety of other saccadic paradigms have been developed in which the role of top-down factors are more pronounced. The remainder of this section will briefly address these tasks.

3.6.1. Delayed prosaccade task

The delayed prosaccade task differs from the standard prosaccade task, as it calls upon greater levels of endogenous processing. In this task, participants are asked to delay making a prosaccade to a suddenly appearing peripheral target until a given cue. It is common for an auditory tone to act as the "go-signal", cueing the participant to make the saccade. The task can still be performed with relative ease, as similar to making a standard prosaccade, the target stimulus remains visible and acts as the goal location. Previous research has found that participants take longer to saccade to the target in a delayed prosaccade task, compared to a standard prosaccade task (Taylor & Hutton, 2009) and that spatial accuracy is decreased in the delayed prosaccade task (Mosimann et al., 2004). The increase in correct latencies observed in delayed prosaccade tasks compared to standard prosaccade tasks is believed to occur because of the added requirement of interpreting the go signal. Interpreting the go signal requires endogenous processing which adds time to the latency of the prosaccade, whereas in a standard

prosaccade task, latencies will be faster because the appearance of the target stimulus triggers the saccade.

3.6.2. Memory-guided saccade task

Another saccadic task in which the role of endogenous factors is made more explicit is the memory-guided saccade task. In this task, participants are required to look to a central fixation point, then to make a saccade to a location cued by a target stimulus. However, the target stimulus is only briefly presented and a saccade to its location can only be made after a go-signal is given. Although very similar to the delayed prosaccade task, the memory-guided saccade task is more difficult, as the 'to be looked at target' does not remain on screen, meaning the spatial location has to be stored and the subsequent saccade made on the basis of this stored information, rather than exogenous information. The task itself may contain several possible goal locations arranged in a circle, or alternatively, the goal location may appear on a vertical or horizontal line. Just as with the delayed prosaccade task, the memory-guided saccade task also measures distractibility, as participants are required to avoid making prosaccades towards the target when it appears. In addition, successful performance on this task can only be achieved if the spatial location of the goal location is remembered over time, and this requires top-down effort.

Importantly, memory-guided saccades are more likely to show decreased peak velocities compared to visually guided saccades such as standard prosaccades, (Krappman, Everling, & Flohr, 1998). Studies have also found that correct latencies are generally increased in memory-guided saccades compared to visually guided saccades (e.g. White, Sparks, & Stanford, 1994). Another important difference between memory-guided saccades and prosaccades is that the former tends to be less spatially accurate (Becker & Fuchs, 1969). However, experimental conditions may play a role in participants' performance on the memory-guided saccade task. For example, in a study by Ohtsuka, Sawa, & Takeda (1989), participants were tested under dark conditions. As a result, memory-guided saccades tended to overshoot the target. Experimenters need to apply a degree of caution when choosing the experimental conditions for a saccadic task. Normally, the interval between the disappearance of the target and the time at which a saccade must be made to the remembered location is relatively short (typically 1-5 seconds) allowing for the information to be stored in short term or working memory. However, previous research has also shown that using longer intervals, such as

30 seconds, can actually increase the accuracy of saccades to the ‘to be remembered location’ (Ploner, Gaymard, Rivaud, Agid, & Pierrot-Deseilligny, 1998), compared to shorter durations, suggesting that long term memory can be utilized to create spatial locations of the ‘to be remembered’ goal location.

3.6.3. Predictive saccade task

In a predictive saccade task, participants are shown a target stimulus that alternates between two locations (e.g. 10degs to the right and 10degs to the left of centre). After 3-4 alternations, participants begin to make predictive saccades which are initiated ahead of the target appearing in the new location. These types of saccades are believed to be centrally guided, as they are not visually guided by a target, hence they depend on an internal model of the target’s movement (Simo, Kriskey, & Sweeney, 2005). Predictive saccades use spatial working memory resources and performance on this task has been found to be worse in populations that are believed to have working memory deficits, such as patients with Parkinson’s disease and schizophrenia (Hutton et al., 2001; O’Sullivan et al., 1997).

Bronstein & Kennard (1987) defined predictive saccades as any eye movement that occurred less than 100msec before onset of the target stimulus. They found that predictive saccades were much more hypometric than non-predictive saccades, a finding that was later supported (Hutton et al., 2001). Endogenous factors may play a greater role in this task compared to a prosaccade task, because an internal representation of what is required is needed for making predictive saccades.

Part 4 – The Antisaccade Task

4.1. Antisaccades

Perhaps the most interesting of the endogenously guided saccade tasks, is the antisaccade task. This task forms the basis of this thesis and this section will consider extensive literature surrounding it. The antisaccade task has been extensively used, often as part of a larger battery of tests to measure cognitive performance in patient populations. In recent years though, researchers have begun to use the antisaccade task

as a tool for measuring goal-directed behaviour in healthy individuals, as will be discussed in the next section.

In the antisaccade task (Hallett, 1978), participants are required to first fixate a central fixation stimulus, and then make a saccade to the opposite location of a suddenly appearing target. Although the concept of making an antisaccade sounds fairly simple, the highly pre-potent response of looking towards a sudden onset target makes the execution of a successful antisaccade difficult, and on around 20% of trials participants typically look at the target before making an antisaccade to the mirror image location (Fischer & Weber, 1992; Smyrnis et al., 2002). On correct trials, participants are able to make a saccade to the mirror-image location of the target stimulus, with no erroneous prosaccade made to the target. The number of erroneous prosaccade errors (also known as antisaccade error rate) is commonly used as the primary measure of antisaccade performance.

Additional measures of antisaccade performance are sometimes reported, including the average latency of correct antisaccades (i.e. the time it takes to look to the opposite location of the target, without glancing at the target), average error latency (i.e. the time it takes to make an incorrect prosaccade to the target), spatial accuracy and the peak velocity of antisaccades and incorrect prosaccades. In the antisaccade task, the target stimulus normally appears to the left or right of the central fixation point, but as is the case with variations of the prosaccade task, the target could appear above or below the central point (Goldring & Fischer, 1997) or there could be several goal locations or different paradigms (i.e. gap, overlap, see section 3.4).

4.2. Basic findings

4.2.1. Error rate

In the original study, Hallett (1978) reported that participants were able to successfully look to the side opposite to the stimulus. Importantly, he also found that, without training the variability of antisaccade error rate across participants was large (30-80%). However, with training this variability was reduced to between 5-7%. In healthy participants nearly all errors are followed by one or more corrective saccades that take the eye away from the target and towards the goal location (mirror image location) (e.g. Tatler & Hutton, 2007). Previous research has found evidence that antisaccade error

rate does not linearly increase with age. Errors are highest during childhood, but decrease for early adulthood. Subsequently, errors increase gradually through adulthood but then increase dramatically around the age of 60 (Fischer, Biscaldi, & Gezeck, 1997; Klein & Foerster, 2001).

Although most studies report average antisaccade error rates of around 20-25% in healthy participants (see Everling & Fischer, 1998 for review) actual error rates vary greatly across individuals. For example, in the large scale study of 2006 Greek conscripts (Evdokimidis et al., 2002) average error rate was 23%, and varied between 0-100%. In addition, in a sub sample of 947 conscripts, Smyrnis et al. (2002) found that antisaccade error rate varied from 0-100%. Large ranges are also often observed in much smaller scale studies (e.g. 0-91%, Taylor & Hutton, 2007).

This high level of variability is an important topic for research, not least because increased antisaccade error rate has been proposed as a "marker of genetic vulnerability" for schizophrenia (e.g. Calkins, Iacono, & Curtis, 2003; Clementz, McDowell, & Zisook, 1994; McDowell, Myles-Worsley, Coon, Byerley, & Clementz, 1999). A large number of studies have found that antisaccade error rates are increased in patients with schizophrenia, but importantly error rates also appear to be increased in their unaffected relatives (e.g. Ettinger et al., 2004; Karoumi et al., 2001), (see section 4.3.1.). High levels of variance in a healthy population make small differences difficult to detect. In other words, if the standard deviation of antisaccade error rate in a healthy population was low, then a comparatively small increase in error rate in an affected population would be statistically significant. As it is, the large variance in antisaccade error rate means that comparatively large differences in performance between healthy participants and unaffected relatives are needed in order to show a significant difference.

4.2.2. Correct latencies

Typically, correct antisaccade latencies (i.e. the time in msec from target onset to an antisaccade made towards the goal location) are longer than correct prosaccade latencies by around 100msec (e.g. Hutton et al., 1998) although the exact duration can vary greatly across participants (e.g. 160-400msec, Evdokimidis et al., 2002). Presumably, variability in antisaccade latencies across samples can be due to differences in task parameters (see section 4.1), however, differences in task parameters cannot account for within participant variability in antisaccade latencies. Interestingly,

antisaccade error latencies, (i.e. the latency of an erroneous prosaccade to the target) have similar latencies to "standard" prosaccades in the range of 170-210msec (e.g. average error latencies were 208msec in Evdokimidis et al., 2002), but can be as low as 156msec (see Fischer, Gezeck, & Hartnegg 2000). Finally, many studies have found that correct antisaccade latencies are reduced when gap trials (figure C) are used compared to overlap (e.g. Goldring & Fischer, 1997) a finding that is also found with prosaccade latencies (see section 3.4.1.).

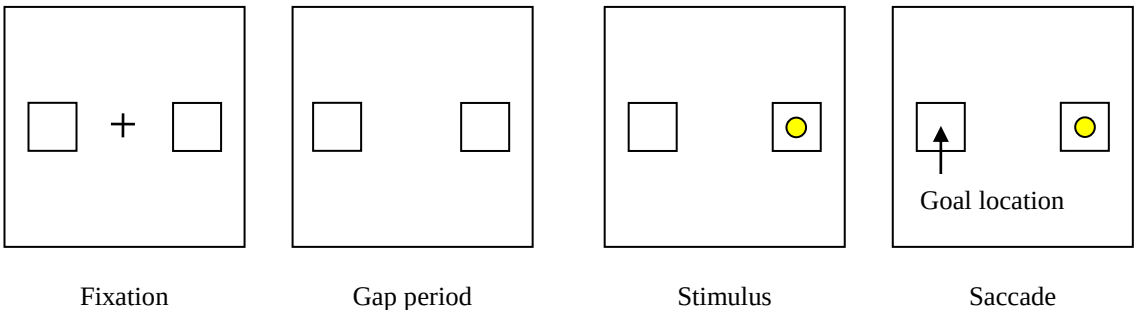

Figure C. Illustration of an antisaccade trial in a gap paradigm

4.2.3. Saccade size

Correct prosaccades are generally less hypometric (undershoot less) than correct antisaccades (Tatler & Hutton, 2007). It is believed that the increased hypometria in an antisaccade is the result of the endogenous requirements associated with making an antisaccade (Edelman, Valenzuela, & Barton, 2006). Specifically, participants are more likely to undershoot the goal location on antisaccades trials because unlike on prosaccade trials, a target stimulus cannot be used as a marker to plot the spatial co-ordinates of the goal location.

4.3. Clinical findings

4.3.1. Schizophrenia

Every published study that has looked at the antisaccade performance in schizophrenia has reported increased antisaccade error rate in schizophrenic patients compared to controls (see Hutton & Ettinger, 2006 for review). Interestingly, deficits to antisaccade performance have been observed in both medicated and unmedicated

schizophrenic patients (e.g. Ettinger et al., 2004). One study observed a significant increase in antisaccade errors for schizophrenic patients who were receiving antipsychotic medication and for drug-naïve patients as well. However, correct latencies were only increased for the drug-naïve schizophrenics (Hutton et al., 1998). Deficits in antisaccade performance shown by schizophrenics are believed to reflect a breakdown of inhibitory mechanisms, mediated by a dysfunctional prefrontal cortex (PFC) (Clementz, 1998).

In order to better understand the schizophrenia phenotype, some researchers have begun to explore behavioural markers (endophenotypes) of the illness. One such behavioural marker is believed to be antisaccade error rate (e.g. Calkins, Curtis, Iacono, & Grove 2004). Support for the idea that antisaccade errors are a marker of genetic vulnerability to Schizophrenia comes from studies that have looked at antisaccade performance in clinically unaffected populations believed to be at risk for the illness. These populations often display similar deficits, (i.e. increased antisaccade errors) to schizophrenic patients. Individuals who display increased levels of schizotypal personality traits (i.e. people with schizotypal personality disorder, or who score highly on schizotypy questionnaires) and unaffected biological relatives of schizophrenia patients are those who are studied the most.

Several studies have found relationships between schizotypal traits and antisaccade performance. An increase in positive schizotypal traits is associated with an increase in antisaccade errors on step trials (e.g. Ettinger et al., 2005; Gooding, 1999; Larrison, Ferrante, Briand, & Sereno, 2000; O'Driscoll, Lenzenweger, & Holzman, 1998; Smyrnis et al., 2003) but not on gap or overlap trials (Holahan & O'Driscoll, 2005; Klein, Brugner, Foerster, Moller, & Schweickhardt, 2000). Similarly, numerous studies have reported increased antisaccade error rate in biological first degree relatives of schizophrenic patients (e.g. Clementz et al., 1994; Curtis, Calkins, Grove, Feil, & Iacono, 2001; Ettinger et al., 2004; McDowell & Clementz, 1997). However, some studies have failed to find increased errors in biological relatives of patients (Brownstein et al., 2003; Crawford et al., 1998; Louchart-de la Chapelle et al., 2005).

4.3.2. Other neuropsychiatric disorders

Some studies have observed increased antisaccade errors in patients with obsessive compulsive disorder (OCD) compared to healthy controls (Rosenberg,

Dick, O'Hearn, & Sweeney, 1997; Tien, Pearlson, Machlin, Bylsma, & Hoehn-Saric, 1992). Although a recent study using more trials found no differences in antisaccade errors or correct latencies between the groups (Spengler et al., 2006). Similarly, no differences in antisaccade errors or correct latencies were found when comparing performance between a sample of patients with attention deficit hyperactivity disorder to healthy controls (Rothlind, Posner, & Schaughency, 1991). However, recent studies have found increased antisaccade errors in children with ADHD (Loe, Feldman, Yasui, & Luna, 2009), increased errors and correct latencies in adolescents with ADHD (Karatekin, 2006) and increased correct latencies in adults with ADHD (Carr, Nigg, & Henderson, 2006).

A recent study showed that compared to healthy controls, patients with fronto-temporal dementia made more antisaccade errors (Meyniel, Rivaud-Pechoux, Damier, & Gaymard, 2005). Similarly increased antisaccade errors were found in a sample of patients with Huntington's disease (Lasker, Zee, Hain, Folstein, & Singer, 1987) and Alzheimer's disease (Abel, Unverzagt, & Yee, 2002). Patients with Parkinson's disease have also showed increased antisaccade errors compared to healthy controls (e.g. Kitagawa, Fukushima, & Tashiro 1994), although some studies have not found differences in errors (e.g. Mosimann et al., 2005). Critically, research using clinical populations may tell us more about the cognitive processes involved with deficits in antisaccade performance.

The studies outlined above highlight the fact that that most neuropsychiatric disorders cause impairments to antisaccade performance. However, despite an extensive literature detailing increased antisaccade errors in clinical populations, it is still not clear exactly why healthy people make antisaccade errors and why some make more errors than others. Specifically, researchers are now beginning to explore possible sources for the often found large variability in antisaccade error rate in healthy participants (e.g. Taylor & Hutton, 2007, 2009).

Whilst the majority of research has investigated antisaccade performance in clinical patients, (e.g. Hutton & Ettinger, 2006), it is still unclear why antisaccade errors are often increased in neuropsychiatric populations. It would therefore be beneficial to try to understand the cognitive underpinnings of correct antisaccade performance in healthy participants in order to gain a more sophisticated understanding of why various patients show impaired antisaccade performance. Research into the cognitive processes involved in saccadic eye movements in healthy participants will be fundamental in

developing sophisticated models of the neurocognitive processes underlying the ongoing control of purposeful behaviour, and how these processes may become dysfunctional in psychiatric patients.

4.4. Models of antisaccade performance

4.4.1. Two stage model

Earlier accounts of antisaccade performance suggested that a successful antisaccade required two separate processes (Everling & Fischer, 1998; Hallett & Adams, 1980). Firstly, a cancellation signal must be sent to inhibit the prosaccade motor program that is automatically triggered at target onset. Secondly, a new program must be written that generates a volitional antisaccade to the opposite side of the target. An antisaccade error is believed to occur if there is a failure to inhibit the prosaccade motor program (e.g. Everling & Fischer, 1998). Most accounts of increased antisaccade error rate in clinical populations tend to argue that they reflect a frontally mediated failure to inhibit the prepotent prosaccade (see Hutton & Ettinger for a fuller discussion).

4.4.2. Parallel programming model ('race model')

A more recent account of antisaccade performance is the 'parallel programming model', or 'race model' as it is often referred to (Massen, 2004; Munoz & Everling, 2004; Reuter & Kathmann, 2004). Parallel programming accounts of antisaccade performance share some similarities with the LATER model of saccade generation (Carpenter, 1981). As mentioned earlier, the LATER model assumes that at target onset levels of activity in saccade generating neurons begin to rise at a uniform rate until the threshold for triggering a saccade is reached. Drawing on accumulator models of saccade generation (see Carpenter 1981; Carpenter & Williams, 1995; Hanes & Schall, 1996) the parallel processing model assumes that at target onset, a race ensues between neural activity in the prosaccade (exogenous) pathway and in the antisaccade (endogenous) pathway, with the winner reaching the threshold appropriate for triggering the saccade first. In other words, if activity in the exogenous pathway reaches threshold first, then an erroneous prosaccade towards the target will be made first, but if activity in the endogenous pathway reaches threshold first, then a correct antisaccade will be made to the opposite side. Therefore, the faster a correct antisaccade can be

programmed, the more likely it is to win the race, and be initiated before the incorrect prosaccade towards the target. Importantly, an incorrect prosaccade is often closely followed by a corrective antisaccade but if an antisaccade is made first, a prosaccade would not follow. This is because in correct trials, activity supporting the antisaccade reaches threshold first, thus "winning" the competition and the build up of activity supporting the erroneous prosaccade towards the target ceases. Figure D illustrates what happens during the time course of a correct antisaccade trial and an incorrect antisaccade trial respectively and shows the neural representation of the race model for a correct and incorrect antisaccade.

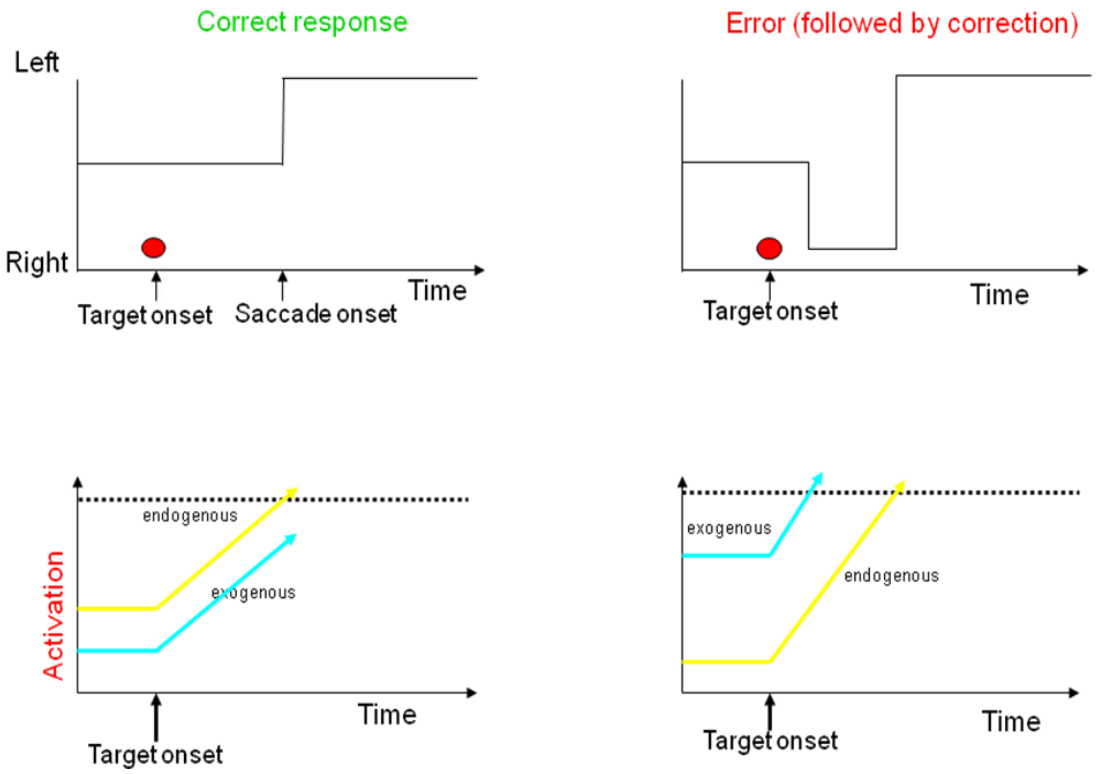

Figure D. The time-course for a correct (top left) and incorrect antisaccade (top right), and the neural representation of the race model for a correct (bottom left) and incorrect antisaccade (bottom right).

Note: in the bottom left diagram, the endogenous pathway supports an antisaccade to the opposite location of the target, and the exogenous pathway supports a prosaccade to the target. In the bottom right diagram, the endogenous program reaches the threshold for saccade triggering, shortly after the error exogenous program gets there, suggesting a corrective antisaccade was made.

According to race model accounts, there is a close relationship between correct antisaccade latency and antisaccade error rate. Any manipulation that differentially affects pro and antisaccade latencies, will ultimately impact on the probability of an antisaccade error being made, as the manipulation will influence the likelihood of one or other of these processes reaching the threshold for saccade triggering first. Conversely, a manipulation that affects prosaccade and antisaccade activity to the same degree, should not impact on antisaccade error rate, as the relative likelihood of either prosaccade or antisaccade neural activity reaching threshold first remains unchanged. In a series of experiments, Massen (2004) tested the predictions of the 'race model.' In her first experiment, she compared performance in separate blocks of pro or antisaccades with performance on a mixed pro/antisaccade paradigm in which participants were unable to predict the upcoming saccade on the basis of the previous saccade. The reasoning was that in the mixed block, participants would not be able to predict the upcoming antisaccade (because each trial could be either a prosaccade or antisaccade), so therefore they should take longer to make correct antisaccades, and as a result, antisaccade errors should increase (as the activity in the prosaccade pathway would be more likely to reach the threshold for saccade triggering first). As predicted, correct antisaccade latencies were increased in the mixed paradigm compared to antisaccade latencies in the separate block of antisaccades, as was antisaccade error rate.

In her second experiment, Massen (2004) attempted to exploit the fact that many individuals show asymmetries in antisaccade performance, making more errors when the target appears on one side compared to the other. Based on the findings of Fischer & Weber (1997), Massen predicted that participants' prosaccade latencies would be faster to the side that they made most errors to, or that correct antisaccade latencies would be slower to the other side. In other words, if a participant made more antisaccade errors to the left side, then their prosaccade latencies should be faster to the left side (compared to the right) or antisaccade latencies are slower to the right side (or both). A second prediction was that prosaccade latencies should be faster or antisaccade latencies should be slower (or both) to targets positioned at 12° compared to 6°. The results showed that on average participants made more antisaccade errors to the left than to the right; however, there was no difference in errors between the different eccentricities. In addition, there was no difference in correct antisaccade latencies when comparing left to right, but antisaccade latencies were faster when the target was presented at 12°.

The second experiment designed to exploit asymmetries in antisaccade performance showed only moderate support for parallel processing predictions, as participants with side asymmetries in antisaccade error rate showed shorter prosaccade latencies to the side where many antisaccade errors were made and slower antisaccade latencies to the opposite side. Although varying stimulus eccentricity did not impact on antisaccade error rate, the effect of this manipulation was consistent with parallel programming predictions. As mentioned above, parallel programming models predict that a manipulation which affects prosaccade and antisaccade latencies to the same degree should not result in a change in antisaccade errors because the likelihood of either prosaccade or antisaccade neural activity reaching the threshold for triggering a saccade first remains unchanged. The results of experiment 2 are in line with this prediction, as prosaccade and antisaccade latencies were both reduced by an increase in stimulus eccentricity.

In the final experiment, Massen measured the effect of inhibition of return (IOR) on antisaccade performance. As mentioned in section 3, IOR is defined as reduced attentional priority for information in a region that has recently experienced a higher priority. Its effect is to bias attentional orienting away from previously inspected locations. Massen used exogenous and endogenous cues to see if IOR would impact on antisaccade error rate. In the first part of the experiment, participants performed separate blocks of prosaccades and antisaccades After fixating a central cross, a cue (white asterix) was presented for 300msec at either the location of the upcoming target (cued trials) the opposite location or at the fixation location (neutral). The cue was followed by a gap of 200msec. In order to draw attention back to the central fixation point, the cue reappeared in the centre, replacing the central fixation cross for another 300msec. Then the central fixation cross reappeared for 200msec, and the target stimulus (green circle) was presented either in the centre of the screen, or in one of the flanker boxes to the left or right of centre. In the second part of the final experiment, centrally presented arrows (endogenous cues) were used to cue attention rather than the asterix.

For part one of the experiment, based on the findings of Rafal, Egly, & Rhodes (1994), Massen predicted that if exogenous cues are able to induce IOR, then detection of the stimulus will be slowed. This means that prosaccade and antisaccade latencies will be affected to a similar degree, thus antisaccade errors should be similar in the trials with uncued and cued stimulus presentation. For the second part, Massen reasoned that if endogenous cues (central arrows) induce IOR, then prosaccade latencies should be

slower when trials are correctly cued compared to when the cue is opposite to the target stimulus location whereas antisaccade latencies should be about the same. This would mean a reduction in antisaccade error rate because the exogenous component in the antisaccade task is selectively slowed allowing activity in the antisaccade pathway to reach the threshold for saccade triggering first in the cued condition, compared to in the condition where the cue does not match the target stimulus location. Essentially, activity in the exogenous (prosaccade) pathway is slowed in the condition with cued stimulus, which should lead to reduced antisaccade error rate in this condition.

The results for part one showed that prosaccade and correct antisaccade latencies were slower when the cue was presented at the same location as the target, compared to when the opposite side of the target was cued. There was no effect of cue condition on antisaccade error rate; therefore errors were equivalent between the cue condition and the uncued condition. As predicted, because both pro and antisaccade latencies were affected equally, this resulted in no change to antisaccade error rate. This finding fits into competitive race model predictions.

In part two, prosaccade latencies were longer in the cued condition, compared to when the opposite side of the target was cued, but this effect was considerably smaller for antisaccades. Therefore, antisaccade errors should be reduced in the cued condition, because prosaccade and antisaccade latencies have been affected to a different degree. Specifically, the exogenous component has been slowed, increasing the likelihood that activity in the endogenous pathway (antisaccade) will reach the threshold for triggering a saccade first. Further analysis showed that as expected, antisaccade errors were reduced in the cued condition.

A recent study by Reuter, Herzog, & Kathmann (2006) provides further support for the race models of antisaccade performance. They found that both schizophrenic patients and healthy participants made more errors and were slower to make antisaccades when a cue had been presented to the side opposite to the target, compared to when no cue was given. This finding conforms to the predictions of race model accounts of antisaccade performance and emphasises the close relationship between speed and accuracy that these accounts have put forward. These findings support race model predictions, as these accounts suggest that an increase in antisaccade latencies will mean an increase in antisaccade errors. However, it is difficult to appropriately test the predictions of the race model without the same manipulation being used on prosaccade performance.

The results of Massen's study are important, as they show that manipulations resulting in increased correct antisaccade latencies (exp. 1) also result in increased antisaccade errors, confirming race model predictions that if the exogenous prosaccade is slowed, there is a greater probability of the endogenously triggered antisaccade winning the competition and reaching the threshold for triggering a saccade first. These findings also highlight that manipulations that affect prosaccade and antisaccade latencies to the same degree, will not impact on antisaccade errors (exp. 2), as both pathways have been influenced, which has no bearing on the race to saccade triggering.

4.4.3. Further evidence of parallel programming

Further support for the idea that exogenous and endogenous signals are programmed in parallel is provided by studies that use oculomotor capture paradigms (e.g. Godjin & Theeuwes, 2002; Theeuwes, Kramer, Hahn & Irwin 1998). In these studies, participants have to make a saccade to a colour singleton, while ignoring the sudden onset of a distracter stimulus. This task shares similarities to the antisaccade task, as in both tasks, an endogenous saccade is programmed (saccade to colour singleton/saccade to mirror image location of target) in competition with an exogenous saccade (saccade to sudden onset distracter/saccade to target). Theeuwes, Kramer,Hahn, Irwin, & Zelinsky (1999) required participants to move their eyes to a uniquely coloured grey circle (target) within a set of six red circles (each containing premasks, i.e. a character disguising another character) and to determine whether the letter inside the target was a C or a reversed C. After 1000msec, every circle except the target circle changed from grey to red and the premasks changed into small letters. On 50% of trials, an additional red circle (onset distractor) was added, the same time as the circles changed colour. Results showed that on about 33% of all trials, participants made an initial saccade to the onset distractor, even though it was irrelevant to the task goal. In addition, durations of fixations on the distractor were in the region of 150msec. According to the authors, these durations were too short to allow the programming of a new saccade.

Theeuwes et al. (1999) suggested that two saccade programs (an exogenous saccade towards the distractor and an endogenous saccade towards the target) were initiated and completed, and the program that was completed first, was the one that was executed first. This model, termed the 'independent horse race model' bears a strong resemblance to current competitive race model accounts of antisaccade performance as

detailed above. Both race model accounts of antisaccade performance and independent horse race accounts of oculomotor capture performance suggest that the two saccade programs are independent of one another, and the destination of the first saccade depends on which program is completed first.

Although there is agreement that separate systems exist for the programming of exogenous and endogenous saccades, the location of where these programs compete with each other remains a topic of debate. For instance, based on research that has shown the involvement of a posterior pathway projecting to the superior colliculus (SC) and an anterior pathway involving the frontal eye fields (FEF) in the programming of saccades, Theeuwes et al. (1998, 1999) suggested that exogenous saccades might be programmed in the posterior pathway and endogenous saccades in the anterior pathway. This suggestion is plausible, given the involvement of the SC in exogenous programming and the FEF in the triggering of intentional saccades (see figure B).

Other advocates of competitive race accounts suggest that the race between exogenous and endogenous signals takes place in the intermediate layers of the SC (Godjin & Theeuwes 2002; Hunt, Olk, Muhlenen, & Kingstone, 2004; Munoz, Dorris, Pare, & Everling, 2000; Trappenberg, Dorris, Munoz, & Klein, 2001). These competitive integration accounts assume that the control signals for exogenous and endogenous saccades congregate on a communal saccade map. Therefore, the programming of the saccade is determined by competing activation at different locations in the saccade map. Competitive integration accounts suggest that when two nearby locations are activated, the combined activation results in a relatively high peak somewhere between the two locations. This idea is similar to the ‘centre of gravity effect’ proposed by Findlay (1992) (see section 3.4.3). Again, competitive integration models are plausible, given that the SC is believed to use saccadic motor maps to determine the direction and size of a saccade (see section 2.2).

One criticism of parallel programming accounts is they assume the race between activity in the prosaccade and antisaccade pathways begins at target onset. This may be true on most trials, but research suggests that incorrect antisaccades are not always followed by a correct antisaccade, on a small percentage of trials the error is followed by one (or sometimes more) saccades that continue to move the eye towards the target (Tatler & Hutton, 2007). In these trials it appears that the intention to generate a correct antisaccade may not necessarily have been in place at the time of target onset, and therefore activity may not have begun to increase in the antisaccade pathway until after

the error had already been made. The extent to which antisaccade performance in healthy participants can be accommodated by predictions of these accounts remains under researched. One aim of this thesis is to systematically test the predictions of competitive race models of antisaccade performance.

4.4.4. Goal activation account

Another popular account of antisaccade performance is the 'goal activation hypothesis'. Goal activation accounts place emphasis on the importance of working memory in the antisaccade task (de Jong, 2001; Eenshuistra, Ridderinkhof, & van der Molen, 2004; Nieuwenhuis, Broerse, Nielen, & de Jong, 2004; Reuter & Kathmann, 2004). Nieuwenhuis et al. (2004) describe 'goal activation' as "turning a task requirement into an appropriate goal and maintaining this goal over time and doing so in the face of competing response tendencies". Critically, it is argued that those participants who show good goal activation will be able to maintain task-relevant information whilst ignoring task-irrelevant information.

The goal activation hypothesis suggests that the ability to make an accurate antisaccade depends largely on the ability to activate and maintain the task goal within working memory. In other words, if task relevant information (e.g. the intention to make antisaccade), is adequately maintained then a prosaccade error should be avoided. It is not sufficient to simply understand the antisaccade task instructions, on any given antisaccade trial, a participant may be aware that they have to avoid looking at the impending target and saccade to the opposite location, the critical point is that they maintain a sufficient level of goal activation to ensure that the intention is carried out. Importantly, activating the task goal and maintaining the task goal can be mediated by several factors, such as concurrent task requirements, task instructions, and environmental structure (Nieuwenhuis et al., 2004).

As discussed earlier, advocates of the two stage model of antisaccade performance (Everling & Fischer, 1998; Hallet & Adams, 1980) suggested that in order to make an accurate antisaccade, an automatic prosaccade towards the target has to be cancelled and a re-directed antisaccade to the opposite location has to be programmed. Therefore they made the distinction that the prosaccade had to be inhibited before the antisaccade was initiated. The goal activation hypothesis on the other hand suggests that inhibition of the prosaccade occurs as a direct consequence of activating the task goal sufficiently (Roberts, Hager, & Heron, 1994). Thus a key difference between the two

stage model and the goal activation hypothesis is that the two stage model suggests an antisaccade error occurs because of a failure to inhibit the incorrect response, whereas the goal activation hypothesis suggests antisaccade errors result from a failure to sufficiently activate the correct response.

In a recent paper, Nieuwenhuis et al. (2004) presented findings from earlier published papers to support a goal activation account of antisaccade performance. This theoretical paper highlights the importance of goal activation and goal neglect in antisaccade performance and to executive functioning in general. Goal neglect is a failure to appropriately maintain the task goal and is believed to be a central element of executive dysfunction (Duncan, 1995). In their first reported experiment, a group of young and a group of older adults performed prosaccades and antisaccades. Stimuli consisted of four boxes situated around the central fixation stimulus (above, below, left right). Participants fixated on the central point, and then one of the four boxes briefly disappeared then reappeared, acting as a cue for the upcoming saccade. After a variable stimulus onset asynchrony (SOA), between 100-1500msec, the target stimulus (a happy or sad schematic face) was presented either in the box that was cued or opposite to the cued box. The cue was termed a pro-cue if it appeared in the same box as the target and an anti-cue if it appeared in the opposite box. Participants then made either a prosaccade to the target, or an antisaccade to the opposite location of the target depending on the block. Prosaccades and antisaccades were performed in separate blocks, but each block included neutral trials, where all four boxes were cued. This meant that cue information could not indicate the location of the upcoming saccade. In addition, participants' were required to identify the emotion of the schematic face target.

Nieuwenhuis et al. argued that the accuracy of target identification associated with the additional choice response task (i.e. identifying emotion of schematic face), would only be possible if participants' saccade to the correct location. The authors assumed that the time needed to (overtly) attend to a certain location can be inferred from the accuracy of identifying targets at that location at various points in time, providing SOA's are manipulated. They predicted that long SOA's (e.g. > 1s) should allow for a higher chance of target identification, because they allow potential saccade errors to be corrected in time. Short SOA's should result in poorer identification of the target because the eyes will arrive too late to foveate the target. The results showed that the young group made less prosaccade and antisaccade errors compared to the older group and when collapsed over age, more antisaccade errors were made compared to

prosaccade errors. As expected, longer SOA's resulted in higher accuracy (better target identification).

The results of this study were compared to their previous study (Nieuwenhuis, Ridderinkhof, de Jong, kok, & van der Molen, 2000) and were almost identical. However, in their previous study, there was no substantial difference between levels of accuracy in the pro-cue and anti-cue conditions for older adults. This suggests that explicitly telling participants to make active use of the cue by means of a saccade toward the target location (as was the case in Nieuwenhuis et al. 2004) prior to the start of the experimental block, reinforced the task goal, meaning older adults were better able to maintain the task goal within working memory and ultimately perform better than if they were not given explicit instructions. This finding highlights the importance of task instructions to goal activation, as outlined by Nieuwenhuis et al. and will be addressed in more detail in paper 3 of this thesis.

The finding that older adults made more antisaccade errors compared to young adults suggests that with increasing age, activation and maintenance of the task goal becomes more difficult. This inability to sufficiently activate the internal representation of the task goal has been found in other populations who demonstrate working memory deficiencies, e.g. Schizophrenic patients (e.g. Reuter & Kathmann, 2004) and this was the motivation behind Nieuwenhuis et al's second presented study.

In their second experiment, Nieuwenhuis et al. (2004) asked whether deficits in goal activation, or 'goal neglect', could account for poor antisaccade performance in schizophrenic patients. A plethora of studies have found that patients with schizophrenia make more antisaccade errors compared to healthy controls (see Hutton & Ettinger, 2006). This finding is often attributed to deficits in inhibitory control mediated by a dysfunctional prefrontal cortex (e.g. Clementz, 1998), a cortical area highly associated with deficits in goal activation (Duncan et al., 2000). The authors argued that if schizophrenia is characterised by a goal activation deficiency, then antisaccade performance should be impaired in schizophrenic patients compared to healthy controls. The stimuli, design and procedure were the same as in their first experiment, except that SOAs were 200, 600 or 1400msec. Overall, healthy controls made less prosaccade and antisaccade errors compared to the patients with schizophrenia. Healthy controls were more accurate as SOA increased, however this was more pronounced for antisaccades than prosaccades. The authors suggest that this

result occurs as, when given more time between cue and target, healthy participants are better able to activate the task goal (appropriately maintain the intention to antisaccade) within working memory, consequently making less antisaccade errors. However, the fact that this increase in SOA was not beneficial for schizophrenic patients (in terms of reducing errors), suggests that even when Schizophrenics are given more time to activate the task goal, they fail to use this to their advantage. This supports the idea that schizophrenia is characterised by a goal activation deficiency. Further support for the idea that schizophrenic patients have impaired goal activation was shown by the fact that they failed to use experimenter instructions to use the cues to reduce errors.

These results suggest that one important aspect of antisaccade performance variability may be attributed to failures to act upon the instructions at the appropriate moment. Therefore, the importance of maintaining the task goal for successful antisaccade performance may depend, in part, on whether the task instructions are appropriately used to mediate goal activation within working memory. The use of task instructions as a mediator of goal activation in antisaccade performance will be investigated in paper 3 of this thesis.

Duncan et al. (2000) argue that goal activation is associated with the prefrontal cortex, and have shown that populations with dysfunction in the lateral prefrontal cortex experience significant deficits in goal activation. Therefore in their third and final experiment, Nieuwenhuis et al. (2004) wanted to see if goal activation deficits were found in other patients whose dysfunction is not restricted to the lateral prefrontal cortex. Using the same design as the previous experiments, they now compared prosaccade and antisaccade performance between healthy controls and patients with obsessive compulsive disorder (OCD), a disorder associated with orbito-medial regions of the prefrontal cortex (Swedo et al., 1989). The authors predicted that OCD patients and healthy controls would make a similar amount of errors on the antisaccade identification task because the disorder is not associated with dysfunctional lateral pre-frontal cortex. The results showed that there were indeed no differences in prosaccade or antisaccade errors made between healthy controls and OCD patients. This finding gives support to the idea that goal neglect is specifically characteristic of neuropsychological populations with dysfunction of the lateral prefrontal cortex (i.e. not OCD patients). Taken together, Nieuwenhuis et al. argue that these findings emphasise the importance of task instructions, environmental structure and concurrent task

requirements to maintaining the appropriate level of goal activation for making antisaccades.

It is, however, difficult to make direct comparisons between antisaccade performance in the experiments reported in Nieuwenhuis et al. (2004) and antisaccade performance in other studies, because in their experiments, the goal of making antisaccades was subordinate to the actual goal of identifying the target stimulus. In other words, participants were not given explicit instructions to make antisaccades. This may have impacted on performance, and meant it was more difficult to uphold an appropriate level of goal activation. Again, this highlights the importance of task instructions, as a mediator of goal activation in antisaccade performance.

4.4.5. Activating the task goal over time

The evidence from Nieuwenhuis et al. (2004) is important because it provides support for goal activation accounts of antisaccade performance. Presumably, the ability to activate and appropriately maintain the task goal builds up over time. If this is the case, then the more time a participant has to prepare the antisaccade (i.e. the longer the interval between the offset of the instruction cue which indicates whether a pro or antisaccade should be made, to the onset of the visual cue or target stimulus) the more time they have to activate the task goal within working memory and in turn the more likely it is they will avoid making an erroneous prosaccade to the target. This hypothesis formed part of the rationale of the 1st paper of this thesis. Several studies have found that antisaccade errors are reduced when the interval between the instruction cue and target (cue lead time - CLT) is increased (e.g. Nieuwenhuis et al., 2004; Weber, Durr, & Fischer, 1998), compared to when it is short. The amount of CLT also impacts on correct antisaccade latencies, particularly in mixed pro/antisaccade experiments, where participants are presented with interleaved prosaccades and antisaccades. For example, when an antisaccade was preceded by a prosaccade, Barton, Greenzang, Hefter, Edelman, & Manoach (2006) observed reduced antisaccade latencies if the CLT was long (2000msec) and an increase in antisaccade latencies if the CLT was short (200msec). Again, it is plausible that a longer preparatory interval allows more time for the task goal to be properly activated and maintained.

In order to test whether a longer preparation interval will result in reduced antisaccade error rate, a mixed pro/antisaccade design will be used in the 1st paper of this thesis. In a mixed design, it is possible to use an instruction cue to indicate to

participants whether they are to perform a prosaccade or an antisaccade. If instruction lead time is manipulated (i.e. the amount of time in msec from the offset of the instruction cue to the onset of the actual cue), then we can see if a longer preparation interval results in improved antisaccade performance. Previous research has found increased antisaccade errors and correct latencies when participants performed mixed pro/antisaccade blocks of trials compared to pure blocks of antisaccades (e.g. Ethridge, Brambhatt, Gao, McDowell, & Clementz, 2009; Olk & Kingstone, 2003). According to Unsworth et al. (2004) the attentional demands of mixed blocks are greater, as the task set has to be switched continually over trials. They argue that in a mixed block, on antisaccade trials, it is harder to actively maintain the appropriate task goal (make an antisaccade), as the upcoming trial is not predicted by the previous trial, thus resulting in increased antisaccade errors and latencies. Using a mixed design in paper 1 of this thesis will almost certainly place demands on the task and make it more difficult for participants to sufficiently activate and maintain the task goal in working memory.

4.4.6. Goal activation and cueing

Goal activation accounts have been useful in explaining other aspects of antisaccade performance in healthy participants. For example, Fischer & Weber (1996) found increased antisaccade errors and latencies when the goal location (opposite side to target) was cued. This somewhat paradoxical finding was later replicated (Weber et al., 1998). According to Fischer & Weber, cueing the correct location for an antisaccade increases error rate because participants adopt a 'mode' or 'task set' depending on which task they have to perform (prosaccade or antisaccade) and treat the cue as a "go signal". Therefore if participants are successful in activating the 'antisaccade task set' when the goal location of the antisaccade is cued it has the unfortunate effect of directing attention to the opposite side (where the target is about to appear) thus increasing capture errors when the target does appear and increasing the latencies of saccades made to the correct location. The idea that participants adopt an antisaccade 'task set' suggested by Fischer & Weber (1996) has clear parallels with goal activation accounts such as those outlined above. Based on this, the 1st paper in this thesis will explore if those who were better able to access the 'antisaccade task set' would be more susceptible to cueing effects.

4.4.7. Goal activation and 'dual task paradigms'

According to goal activation accounts, successful antisaccade performance requires the ability to maintain and manipulate task-relevant information in mind (working memory), whilst simultaneously ignoring task-irrelevant information and over-riding prepotent responses (inhibition). These two functions suggest a close relationship between goal activation and working memory. Some studies that support a goal activation account of antisaccade performance have used 'dual task paradigms' to investigate the role of working memory in antisaccade performance (e.g. Claeys et al., 1999; Mitchell, McCrae, & Gilchrist, 2002; Roberts et al., 1994). In a dual task paradigm, participants perform antisaccades whilst simultaneously performing a concurrent task. In an interesting study, Stuyven, Van der Goten, Vandierendonck, Claeys, & Crevits (2000) investigated the effects of cognitive load on antisaccade performance. The authors wanted to know whether a possible effect of cognitive load on antisaccade performance could be due to a central cognitive component, a motor component, or simply to the fact that two tasks have to be performed at the same time. In their first experiment, participants completed a block of antisaccades, a block of antisaccades with the Random time Interval Generation (RIG) task (Vandierendonck, De Vooght, & Van der Goten, 1998) and a block of antisaccades with a fixed tapping task. The RIG task required participants to tap an unpredictable rhythm on the zero key of the computer keyboard at an average rate of one keystroke per second. Repetition of a pattern was not allowed. The requirement to be random and to avoid automaticity loads the central executive (De Rammelaere, Stuyven, & Vandierendonck, 2001). For the fixed tapping task, participants were instructed to hit zero on the keyboard at a rate of one tap per second. In addition, participants performed a block of prosaccades, a block of prosaccades with the RIG and a block of prosaccades with the fixed tapping task. The fixed tapping task was included as an additional control condition for the RIG as it requires the same motor actions, but presumably does not require as much executive functioning resources. Compared to when antisaccades were performed alone, antisaccade errors and correct antisaccade latencies were increased when antisaccades were performed with fixed tapping. Similarly, errors and latencies were increased when antisaccades were performed with the RIG task. A similar pattern of results was found for prosaccades, except that the fixed tapping task did not alter prosaccade errors, suggesting that cognitive load impacted more on antisaccades. Participants made more antisaccade errors when performing the RIG task compared to fixed tapping, but there

were no differences in latencies between these conditions. The authors argued that more antisaccade errors were made when antisaccades were performed with the RIG task than the fixed tapping task because the RIG task required working memory processes that would otherwise have been devoted to antisaccade performance. In terms of goal activation accounts of antisaccade performance, it would seem that the RIG task disrupted goal activation more than the fixed planning task. This is presumably due to the increased monitoring demands that are required in the RIG task (i.e. constantly checking that the taps are random) compared to the fixed tapping task.

The relationship between goal activation, working memory and antisaccade performance has also been investigated in studies that have explored individual differences in working memory capacity (the ability to maintain memory representations in the face of concurrent processing, distraction, and/or attention shifts Shah & Miyake, 1999) as a possible predictor of antisaccade performance. One common individual difference that has been used in these studies is participants' scores on the operation span task, a measure that indicates whether a participant has high or low working memory capacity (e.g. Kane, Bleckley, Conway, & Engle, 2001; Unsworth et al., 2004). These studies will be discussed in more detail in the 5th paper of this thesis, where an attempt will be made to find potential sources that can account for the large variability in antisaccade performance in healthy participants.

One relatively unexplored area of antisaccade performance is the degree to which a participant's motivation impacts on error rate and correct latencies. Previous studies have addressed this to some extent, by using incentives as a tool with which to measure motivation. Typically, the type of incentive used is monetary reward, and previous research has found mixed results when looking at the effects of monetary reward on antisaccade performance in healthy participants (e.g. Blaukopf & Di Girolamo, 2006; Hardin, Schroth, Pine, & Ernst, 2007; Jazbec et al., 2006). If antisaccade error rate is reduced by monetary reward, then it can be assumed that the incentive increases activation of the task goal, in line with goal activation accounts. The influence of motivation (using incentives) on antisaccade performance will be explored in more detail in the 2nd paper of this thesis.

The preceding discussion has outlined several key issues concerning antisaccade performance that still remain unclear. The most prominent of these issues is that variability in antisaccade error rate can be enormously large and it is unclear why these individual differences in antisaccade performance exist? Additionally, the extent to

which antisaccade performance can be explained by predictions of recent competitive race model and goal activation accounts remains under researched. The following section, will outline the aims and hypotheses of this thesis based on predictions of current models of antisaccade performance.

Part 5 – Thesis Aims

5.1. Aims

The central aim of this thesis is to explore possible determinants of individual differences in antisaccade performance amongst healthy participants. To this end, the studies attempt to establish the extent to which a range of top-down factors can influence antisaccade performance. A secondary aim of the thesis is to establish the extent to which the effects of these top down factors can be explained within current models of antisaccade performance.

5.2. The articles comprising this thesis

This thesis consists of five research articles. Articles 1-4 describe ten behavioural experiments designed to investigate the effects of various manipulations on antisaccade performance in healthy adults. The fifth article contains correlational analyses in which data from some of the experiments described in articles 1-4 are combined with measures of individual differences such as working memory capacity and processing speed.

5.2.1. Article 1

The opening article describes two experiments which employed a mixed pro and antisaccade task, combined with a peripheral cueing manipulation, to explore the role of goal maintenance in antisaccade performance. The two critical manipulations were preparation time (the amount of warning that the current trial would be a pro or antisaccade) and cue location (on the same or opposite side of the upcoming target). Based on recent goal activation accounts of antisaccade performance (e.g. Nieuwenhuis

et al., 2004) it was hypothesised that those who are better able to sufficiently activate and maintain the task goal, will be better able to activate the 'antisaccade set,' but as a consequence, become more susceptible to 'cueing effects', resulting in more antisaccade errors on trials in which the cue appears at the opposite location to the target. It is also expected that altering preparation times will attenuate the 'cueing effect'.

5.2.2. Article 2

The second article investigates the extent to which participants' antisaccade performance is influenced by their motivation, using varying incentives, in order to further understand the role of goal activation in antisaccade performance. Three experiments varied the monetary and non-monetary incentives participants received whilst performing the antisaccade task. Experiment 1a attempted to clear up inconsistencies within the literature as some studies have found an effect of incentive on antisaccade errors and others have not. Experiment 1b was used to tease apart potential confounds of having feedback and a financial reward in the same condition. Finally, experiment 2 was set up to alleviate the influence of large variations in antisaccade error rate within groups found in experiments 1a and 1b, by using a within-participants design. Again, based on goal activation accounts of antisaccade performance, it was hypothesised that incentives will increase activation of the task goal resulting in improved antisaccade performance, as the incentives will motivate participants to perform better, than if no incentives are given.

5.2.3. Article 3

Article three describes three experiments that explore the role of strategic influences on antisaccade performance. The first experiment looks at the impact of different task instructions on antisaccade performance. Experiment two was designed to replicate the antisaccade findings from experiment one and to also see if prosaccade performance is differentially affected by these instructions. Lastly, experiment three was designed to standardise the requirements of certain instructions from the previous experiments. Predictions of current race models of antisaccade performance were tested in all three studies. In line with competitive race model accounts of antisaccade performance (e.g. Massen, 2004) it was hypothesised that if any of the task instructions increased antisaccade latencies, then antisaccade errors would be increased as well, providing the instructions had a differential effect on prosaccade latencies. This

prediction is based on the assumption that manipulations which slow activity in the antisaccade pathway will increase the likelihood of activity in the prosaccade pathway reaching the threshold for triggering an error first.

5.2.4. Article 4

Previous research suggests that participants are unaware of approximately 50% of the errors they make (Mokler & Fischer, 1999), but no research has attempted to establish whether error awareness can be influenced by manipulations such as those described in the previous articles in this thesis, which are known to affect other antisaccade metrics such as error rate and correct latencies. Following on from article three, the first experiment in this article investigated the potential interaction between different task instructions and awareness of antisaccade errors and to see what effect instructions designed to reduce antisaccade errors have on 'aware' and 'unaware' errors. The second experiment deployed a 'dual task' paradigm to see if manipulations capable of increasing antisaccade errors, would differentially affect 'aware' and 'unaware' errors. We predicted that awareness of antisaccade error rate would be modulated by different top-down factors and that these effects would be different for 'aware' and 'unaware' errors.

5.2.5. Article 5

The final article used a correlational approach to determine the extent to which a range of individual differences could account for the large variability in antisaccade error rate often found in healthy participants (e.g. Smyrnis et al., 2002). Across three analyses, antisaccade metrics were correlated with performance on a range of cognitive tasks and personality measures in an attempt to identify individual differences that might help explain some of this variance. Based on previous findings that a relationship between working memory capacity and antisaccade performance exists, the first analysis looked at potential correlations between antisaccade performance and cognitive measures designed to tax different components of working memory. Based on the notion made by competitive race model accounts that speed of processing may be critical to antisaccade performance, the second analysis explored the potential relationship between antisaccade performance and 'speed of processing' measures. The third and final analysis was interested to see if a relationship existed between antisaccade performance and personality measures that can be likened to traits found in

various patient groups who show deficits in antisaccade performance such as schizotypy and impulsivity. As previous research suggests impaired antisaccade performance in schizophrenic patients is mediated by a working memory deficiency (e.g. Reuter & Kathmann, 2004), it was hypothesised that higher schizotypy scores would be associated with higher antisaccade error rates. In addition, we expected that participants with high impulsivity scores will make more antisaccade errors and take longer to make a correct antisaccade than participants with low impulsivity scores.

Research Articles

Article 1 - The Effects of Cueing on Pro and Antisaccade Performance

A revised and abbreviated version of this paper has been published as: Taylor, A. J. G., & Hutton, S. B. (2007). The effects of individual differences on cued antisaccade performance. *Journal of eye movement research, 1,* (1:5), 1-9.

Abstract

In a prosaccade task, cueing the location to which a correct response should be made results in a decrease in saccade latency. If the correct location in an antisaccade task is cued (i.e. mirror image location of the upcoming target), correct latencies and errors are increased. It has been suggested the increase in error rate occurs because participants adopt an 'antisaccade task set' and treat the cue as if it were a target. According to this account, attention is directed to the mirror image location of the cue, which in the case of an antisaccade is the location where the target subsequently appears. The appearance of the target at a location to which attention has been directed increases the probability of a saccade being made to this location. This hypothesis was tested across two experiments using a mixed pro/antisaccade task. In both studies, the target for pro and antisaccades either appeared uncued, preceded by a cue on the same side as the target stimulus, or preceded by a cue on the opposite side of the target. Varying instruction lead times (ILT) of 500 and 2000msec were used in the first study, with 200 and 1000msec used in study 2. Cue lead time remained constant at 100msec throughout. In experiment 1, as predicted, cueing the location of a correct antisaccade had the paradoxical effect of increasing errors but reducing correct latencies. However, no interaction was found between trial type and ILT. In experiment 2 we replicated the paradoxical cueing effect, but again failed to show that having less time to prepare would result in a reduced cueing effect. Overall, there was no difference regarding the cueing effect, between participants who activated the 'antisaccade set' and participants who did not. The findings suggest the visual properties of a cue are used to aid in the decision of the location of the subsequent saccade.

Introduction

Often, in everyday life the execution of a correct behavioural response requires a more habitual over-learned response to be suppressed. For example in order to drive home via a specific shop, we may need to take a different route, and turn right at a junction instead of left which would be the usual route home. Situations like these occasionally produce unintended behaviour known as 'action slips'. Research has found that these slips are likely to occur more often when we are distracted, and are therefore less likely to have sufficiently maintained the task goal in mind (Reason, 1984). A laboratory task that has been used to investigate the cognitive processes underlying action slips is the antisaccade task. In this task participants are required to overcome the strong tendency to saccade towards a sudden onset target and instead make a saccade to the mirror image location. Healthy participants typically make an error (fail to suppress an erroneous prosaccade towards the target), on around 20-25% of trials (e.g. Smyrnis et al., 2002). Numerous studies have shown that compared to healthy controls, antisaccade errors are increased in patients with schizophrenia (e.g. Fukushima et al., 1988; Hutton et al., 1998; Matsue et al., 1994; Katsanis, Kortenkamp, Iacono, & Grove ,1997) and patients with lesions to the dorsolateral prefrontal cortex (e.g. Guitton, Buchtel & Douglas, 1985; Pierrot-Deseilligny et al., 2003). However, despite a large literature detailing antisaccade performance in clinical populations (see Hutton & Ettinger, 2006 for review), it is still not clear exactly why healthy people make antisaccade errors and why some make more errors than others.

According to early 'cancellation models' of antisaccade performance, the sudden appearance of the target automatically triggers a motor program for a prosaccade in its direction. Errors occur when certain endogenous processes fail to inhibit or cancel this program (Hallett & Adams, 1980; Everling & Fischer, 1998). Parallel programming accounts of antisaccade performance (Massen 2004; Munoz & Everling, 2004; Reuter & Kathmann, 2004; Reuter, Rakusan, & Kathmann, 2005) have suggested that a competition exists between competing prosaccade (exogenous) and antisaccade (endogenous) pathways. In other words, at target onset, a 'race' occurs between activation in the neural pathway supporting the exogenous saccade and activation in the neural pathway supporting the endogenous saccade. If activation in the exogenous pathway reaches the threshold for triggering a saccade before activity in the endogenous

pathway reaches the threshold, an erroneous prosaccade is made. Activity continues to rise in the correct endogenous pathway, resulting in a correct antisaccade shortly afterwards. Recent goal activation accounts of antisaccade performance (e.g. Nieuwenhuis, Broerse, Nielen, & de Jong, 2004; Hutton, 2008), have postulated that errors are more likely to occur if the level of goal activation within working memory is insufficient. In other words, if the goal to make a saccade to the mirror image location of the impending target is insufficiently activated, then activity in the endogenous pathway starts from a low baseline, and is therefore less likely to 'win' the race and reach threshold before the activity supporting the exogenous prosaccade.

Previous research on prosaccades has demonstrated that cueing the correct location (i.e. the location at which the target stimulus appears), results in reduced saccade latencies (Fischer & Weber, 1998). This finding is traditionally interpreted as reflecting the effect of attention, the cue serves to shift attention to the target location, resulting in faster target detection when it subsequently appears (Müller 1994; Posner 1980; Remington, 1980). The relationship between eye movements and attention remains a topic of considerable debate (see e.g. Hutton 2008). Fischer & Weber (1993) suggest that visual attention is an important mechanism that is related to saccadic eye movements. They proposed that the attention system has to first 'disengage', then 'move', then 'engage'. Therefore, it is not fixation of a foveal stimulus as such that suppresses the saccade system but rather attention being voluntarily directed to a visual stimulus. This is important, as this implies a loose functional relationship between eye movements and attention. In contrast, the pre-motor theory of attention (Rizzolatti, Riggio, Dascola, & Umilta, 1987) suggests there is a functional relationship between attention and eye movements and the allocation of spatial attention to a specific location is equivalent to planning, but not executing a saccade to that location. The pre-motor theory thus suggests that programming a saccadic eye movement, results in a shift in attention. However, the Visual Attention Model (VAM) (Schneider, 1995) provides a contrasting conclusion. The model suggests that targets are selected by a visual attention mechanism that is responsible for both ''selection for action' and ''selection for perception'. The VAM advocates that a shift in attention will affect the programming of a saccade. However, despite this disagreement in the literature, the neural relationship between attention and eye movements is believed to overlap considerably. For example, Beauchamp, Petit, Ellmore, Ingeholm, & Haxby (2001) found increased neural

activation in several brain areas on both covert (attention without saccade) and overt (attention with saccade) shifts of attention.

Interestingly cuing peripheral locations also impacts on antisaccade performance. Perhaps surprisingly, Fischer & Weber (1996, exp. 1) found increased antisaccade error rates and increased correct antisaccade latencies when they used a block of trials in which the goal location was cued. In their second experiment, they replicated the finding of increased antisaccade error rates and also found reduced correct antisaccade latencies in trials that were cued the same side as the target stimulus (which, in the antisaccade task, is the opposite site to the goal location). In another study, using gap and overlap paradigms, Weber, Dürr, & Fischer (1998) also found that antisaccade errors were increased compared to control trials, when the goal location was cued. Fischer & Weber (1996) suggested that this paradoxical finding occurs because participants adopt a specific 'task set' when performing the pro or antisaccade task and participants treat the cue as if it was a target, or 'go signal'. In the prosaccade task, where a correct response is a saccade towards the target, this results in reduced latencies. In the antisaccade task, however, a correct response must be made to the opposite side of the sudden onset target. If the location to which a correct antisaccade should be directed is cued, participants that have adopted an 'antisaccade task set' may treat the cue as if it were a target, with the unfortunate effect that they direct their attention to the opposite side, where the target is about to appear. If attention is already oriented to the location at which a target then subsequently appears, it stands a greater chance of eliciting a saccade in its direction (thus explaining the increase in antisaccade errors) and participants will also take longer to initiate a correct saccade (because attention has to be reoriented to the opposite side).

The idea that participants adopt an antisaccade 'task set' suggested by Fischer & Weber (1996) has clear parallels with goal activation accounts such as those outlined above. According to a goal activation interpretation, those participants who are most able to successfully adopt an 'antisaccade task set' (or activate the task goal within working memory) should be more vulnerable to the detrimental effects of cueing the correct location in the antisaccade task, as they will be most likely to treat the cue as if it were a target. In other words cueing the correct location should result in a larger decrement in performance in those participants who are in fact best at the task. This prediction was confirmed by Reuter, Herzog & Kathmann (2006) who found that the effect of pre-cuing the goal location was weaker in a group of schizophrenic patients,

compared to healthy controls. If goal activation mediates the detrimental effect of cueing in the antisaccade task, a further prediction would be that the less time available to activate the antisaccade task set, the smaller the detrimental effect of cueing the correct location.

Research into task switching suggests that activating a specific goal or shifting between goals (as occurs on mixed pro / antisaccade tasks) is not instantaneous. For example, Rogers & Monsell (1995) investigated task switching by asking participants to either classify the letter member of a pair of characters as a vowel or consonant, or to classify the digit member of a pair of characters as even or odd. They found a task switching cost, i.e. response time was slower on trials where the task goal was switched (switch trials) than on trials where the task goal remained the same (repeated trials). In another study, Sudevan & Taylor (1987) presented participants with a cue that preceded a digit stimulus that indicated whether this digit had to be classified as odd or even. They also found increased reaction times for switch, compared to non-switch trials, as well as increased error rates. Hodgson, Golding, Molyva, Rosenthal, & Kennard (2004, exp 4.) found switch costs (increased latencies) when they used a mixed block of antisaccades with vertical antisaccades, with the cost being greater when switching from a normal antisaccade to a vertical antisaccade. Taken together, these findings show that having to shift between task goals takes longer than when the task goal remains constant.

In addition to the research demonstrating that switching between task sets requires time, there is an increasing body of evidence that suggests that having activated a specific task set, levels of goal activation can fluctuate over time, with insufficient activation leading to ‘goal neglect’ (Duncan, 1995). These fluctuations in goal activation have been studied within the context of prospective memory tasks (e.g. Duncan, Emslie, Williams, Johnson, & Freer, 1996; Marchant, Trawley, & Rusted, 2008). Successful prospective memory performance requires participants to only resume a task that has been put on hold when a memorized trigger condition is satisfied. Goal neglect has also been found in conflict tasks, where participants have to respond to a set of stimuli, even though more compelling stimuli are obtainable. For example, Milner (1963) found that patients with frontal lobe damage show goal neglect on the Wisconsin card sorting task, because they tend to continue to use old rules that are inappropriate, even though they accept that they should be abandoned. Goal neglect is also relevant to antisaccade performance. For example, in an analysis of contingency effects in

antisaccade performance, Tatler & Hutton (2007) suggested that goal neglect could explain their finding, that the probability of participants making an antisaccade error was increased if an error had been made on the previous trial. Thus goal neglect could be responsible for errors on both the previous trial and current trial, rather than the error on the previous trial causing an error on the current trial. In this instance, goal neglect results in lowered baseline activity, making it more likely that two consecutive errors are made. Together, these findings suggest that the magnitude of the effect of cueing during the antisaccade task may depend on the degree of task activation. By using a mixed pro/antisaccade block, in which the task instruction (pro or antisaccade) is given at the onset of each trial, it is possible to manipulate the amount of time participants have to activate the appropriate 'task set'. If the 'task set' is only weakly activated (as would be expected to occur if the cue appeared very shortly after the instruction to make an antisaccade was given) then the paradoxical impairments in performance that Weber et al. (1998) observed when the goal location was cued might be reduced.

The current study was designed to replicate and extend the findings of Fischer & Weber (1996, 1998) and Weber et al. (1998) by determining the effects of cueing on pro and antisaccade performance and simultaneously manipulating the amount of time participants had to prepare a pro or antisaccade. These previous studies used separate blocks of trials, one containing trials where the cue was always informative of the goal location and one block where the cue was always non informative of the goal location. One problem with this design is that each trial can become predictable, as saccade planning is made easier because the same task set is reused across trials within the block. The present study enhanced and extended this earlier work by including cued trials that were either informative of the goal location, non-informative of the goal location, or uncued trials, all within the same block. By doing this, it was harder for participants to predict the direction of the intended saccade and, in addition, we were able to manipulate the time that participants had to activate the appropriate 'task set' on each trial. In line with goal activation accounts, it was expected that for antisaccades cued to the opposite side to the target, in other words the goal location, would result in an increase in errors, compared to uncued and trials cued the same as the target. We further predicted that the paradoxical effect of cueing should be reduced on trials that participants do not have a long time to prepare for the antisaccade.

Experiment 1

Method

Participants

Participants comprised 58 undergraduate students with normal to corrected normal vision from the University of Sussex, of whom 46 were female and 12 male. Ages ranged from 18 - 31 years (M = 24.6, SD = 3.87). Participants either received course credit or £5 for their participation. Participants were naïve to the purposes of this study. All participants provided written consent, and the experiment was approved by the Department of Psychology Ethics Committee at the University of Sussex.

Apparatus

Eye movements were recorded using an Eyelink II eye tracker (S-R Research Ltd. Ontario Canada) with a temporal resolution of 2msec and a spatial resolution of around 0.25 degs. The stimuli were displayed on a 21inch CRT monitor with a screen resolution of 1,280 x 1,024 pixels and a refresh rate of 100Hz. Actual screen dimensions were 40cm horizontal and 30cm vertical. Participants were seated approximately 70cm from the screen in an adjustable chair that had been modified to prevent any rotational movement. Each set of trials was preceded by a calibration procedure, during which participants focused their eye gaze on 9 separate targets in a 3 x 3 grid. Only right eye movements were recorded.

Stimuli

For each trial the display comprised a black background containing two empty marker boxes placed an equal distance (3.7 cm from their inner edge to the centre of the screen) on the left and right of a yellow central fixation cross. The marker boxes were 1.44 degrees of visual angle in diameter and in white. After 1000msec the central fixation cross was replaced by a colour circle (measuring 0.5 degrees in diameter), which cued the instructions for the present trial. A green circle indicated that a prosaccade was to be made and a red circle indicated that an antisaccade was to be made. The target

comprised a yellow circle (0.25 degrees in diameter) that appeared centred in either of the marker boxes. The target stimulus was displayed for 1500msec, which was enough time for a participant to initiate a saccade towards or away from it. However if a saccade was made that reached the goal location, the target would remain on screen for a further 300msec.

Design/procedures

In a within-participants design, participants performed 192 trials, 96 of which were prosaccades and 96 of which were antisaccades. Prosaccades and antisaccades were interleaved in the same block. After either a short (500msec) or long (2000msec) preparation time (Instruction Lead Time – ILT), the central instruction cue (green for prosaccade or red for antisaccade) was extinguished and a target appeared in one of the flanking marker boxes. On one third of trials the marker box in which the target subsequently appeared was cued by an increase in its width that lasted for 100msec before target onset. These trials were designated Cued Same (CS) trials. In another third of trials the flanking box opposite to the one in which the target subsequently appeared was cued (Cued different - CD trials). The final third of trials were uncued (UC trials). In each instruction / cue combination, an equal number of pro and antisaccade trials were administered, with an equal number of long and short instruction delays. The actual order of trials was randomized for each participant. Twelve practice trials were given first. Participants were not informed about the flashing boxes, or their relevance to cueing.

Measures

We recorded the number of prosaccade errors (saccades made away from the sudden onset target on prosaccade trials), antisaccade errors (saccades made towards the onset target on antisaccade trials) and the latency of correct pro and antisaccade responses. Trials were excluded from analysis if, a) if the eye was not within 40 pixels (approximately 1 degree of visual angle) of the central fixation point at the time of target appearance; b) no saccade was made within the trial duration; c) the primary saccade was obscured by blinks, d) the primary saccade was made < 0msec after target onset, e) the primary saccade was made > 500msec after target onset. These criteria resulted in less than 7% of trials being excluded. Correct prosaccade latency was measured as the difference in milliseconds between target onset and onset of a saccade made towards the

target. Antisaccade latency was measured as the difference in milliseconds between the target onset and the onset of a saccade made to the opposite hemifield (without any intervening erroneous saccade).

Results

Correct prosaccade and antisaccade latencies

A three way 2(task: pro vs. anti) x 3(trial type: UC vs. CS vs. CD) x 2(ILT: 500 vs. 2000) repeated measures analysis of variance, was carried out on correct prosaccade and antisaccade latencies (figure 1.1).

The main effect of task was significant. As is well established, participants were faster to initiate correct prosaccades compared to correct antisaccades ($F(1, 56) = 348.31$, $p < .001$, $r = .93$). There was also a main effect of trial type, $F(1.62, 90.92) = 87.85$, $p < .001$, $r = .70$ (Huynh Feldt, $e = .81$). For both prosaccade and antisaccade trials, cueing the same location as the target resulted in faster latencies compared to no cues (UC) and cueing the opposite location (CD). There was also a main effect of ILT, $F(1, 56) = 20.87$, $p < .001$, $r = .52$, as participants overall were faster to make correct pro and antisaccades when given a longer preparation time (2000 vs. 500msec).

These main effects were qualified by a number of significant interactions, including the interaction between trial type and task ($F(1.55, 86.97) = 22.41$, $p < .001$, $r = .45$, Huynh-Feldt, e = .68), ILT and task $F(1, 56) = 5.62$, $p = .02$, $r = .30$) and the three way interaction between trial type, task and ILT ($F(2, 112) = 5.6$, $p < .01$, $r = .22$, see figure 1.1).

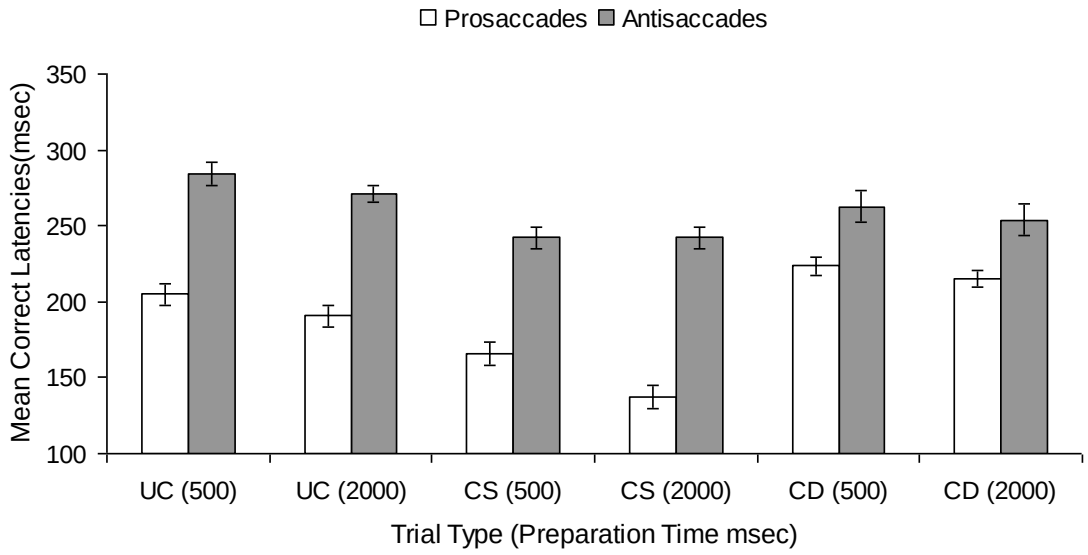

Figure 1.1. Correct saccade latencies across trial type, as a function of task and ILT

The three way interaction was explored with separate 3(trial type) x 2(preparation time) ANOVAs for correct pro and antisaccade latencies.

For the prosaccade trials, the main effect of trial type was significant (F(1.54, 86.04) = 162.69, p < .001, r = .81, Huynh-Feldt, e = .77). Participants were faster to make prosaccades in the CS condition compared to the UC condition (t(57) = 17.17, p < .001, r = .92, and slower to make prosaccades in the CD condition (t(57) = -4.97, p < .001, r = .55), compared to the UC condition. The main effect of trial type was also significant for antisaccade latencies (F(1.53, 87.37) = 13.19, p < .001, r = .36, Huynh Feldt, e = .77). Participants were faster on CS trials compared to UC trials (t(57) = 7.62, p < .001, r = .71). There was a trend for participants to be faster in the CS, compared to CD condition (t(57) = -2.39, p = .06, r = .30). However, participants were not significantly faster in the CD condition, compared to UC condition (t(57) = 2.14, *ns*, r = .27).

The main effect of preparation time was significant for prosaccades F(1, 56) = 29.56, p < .001, r = .59 and the interaction between trial type and preparation time was also significant (F(2, 112) = 8.60, p < .001, r = .27), suggesting that a longer preparation time decreased latencies the most for CS trials (t(57) = 6.75, p < .001, r = .67) followed by UC (t(57) = 3.15, p < .01, r = .38), then CD (t(57) = 2.29, p = .03, r = .29, figure 1.2). For antisaccades, there was also a significant main effect of preparation time (F(1, 57) = 4.84, p = .03, r = .28), as the shorter preparation time resulted in faster

correct latencies. The interaction was non-significant for antisaccades ($F(2, 114) = 2.04$, $p = .13$, $r = .13$, figure 1.3).

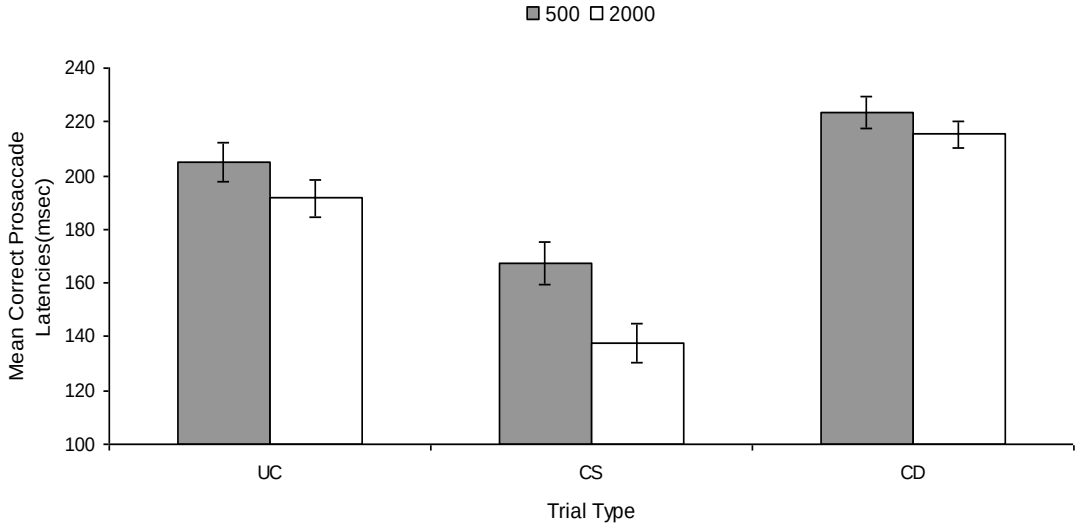

Figure 1.2. Correct prosaccade latencies across trial type, as a function of ILT

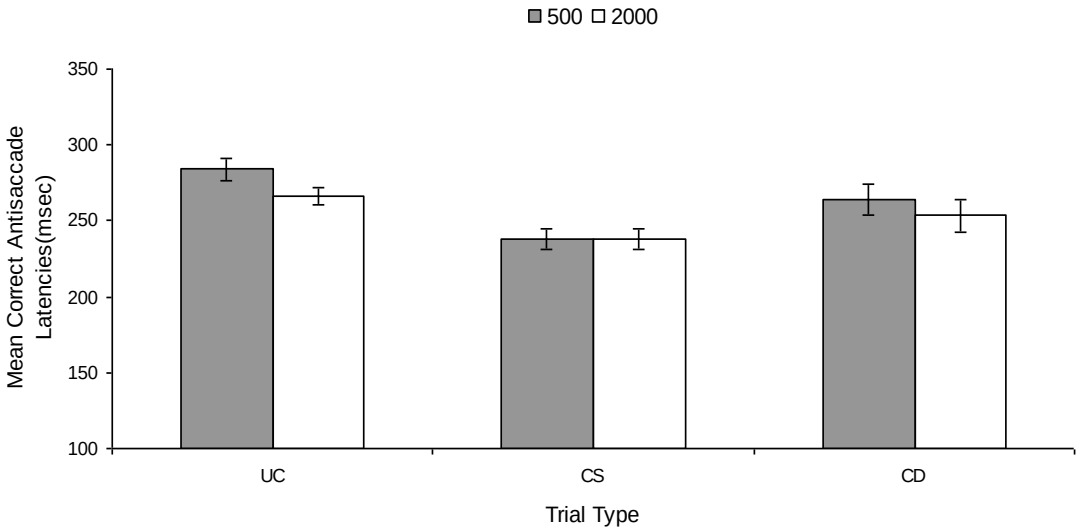

Figure 1.3. Correct antisaccade latencies across trial type, as a function of ILT

Prosaccade and antisaccade errors

A three way 2(task: pro vs. anti) x 3(trial type: UC vs. CS vs. CD) x 2(ILT: 500 vs. 2000) repeated measures analysis of variance, was carried out on prosaccade and antisaccade errors (figure 1.4).

There was a highly significant main effect of task (overall more errors were made in the antisaccade task compared to the prosaccade task $F(1,57) = 107.83$, $p < .001$, $r = .81$). The main effect of trial type was also significant ($F(1.22, 69.24) = 66.67$, $p < .001$, $r = .70$, Huynh-Feldt correction, $e = .61$), with errors being greatest in the CD trials compared to the CS and UC trials. Finally, the main effect of preparation time (ILT) was also significant ($F(1, 57) = 18.16$, $p < .001$, $r = .49$) with more errors being made after the shorter preparation time.

These main effects were qualified by a number of significant interactions. All 2 way interactions and the three way, trial type, by task by ILT interaction were significant (Fs> 9.22, $ps < .03$, $rs > .20$).

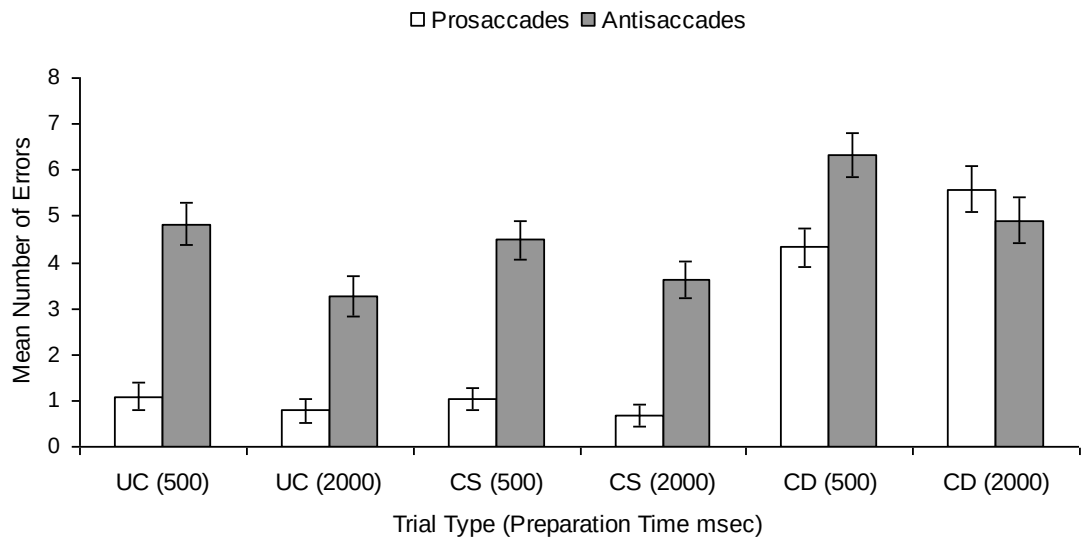

Figure 1.4. Average number of pro and antisaccade errors across trial type as a function of task and ILT

The three way interaction was explored with separate two way, 3(trial type) x 2(preparation time) ANOVAs for pro and antisaccade errors.

The main effects of trial type were significant in both analyses (prosaccades: $F(1.11, 63) = 77.33$, $p < .001$, $r = .74$, Huynh Feldt, $e = .55$; (antisaccades: $F(1.45, 82.75) = 9.23$, $p = .001$, $r = .32$, Huynh Feldt, $e = .73$). For prosaccade errors, paired t-test comparisons revealed there were more errors in the CD trials compared to the UC trials, ($t(57) = -8.89$, $p < .001$, $r = .76$), and on the CD compared to CS trials, ($t(57) = -8.98$, $p < .001$, $r = .77$). There was no difference with prosaccade error rate between UC

and CS trials ($t(57) = .67$, *ns*, $r = .09$). The same pattern was observed for antisaccade errors: paired t-test comparisons revealed that participants made more errors in the CD trials compared to the UC trials ($t(57) = -3.29$, $p < .01$, $r = .39$) and the CS trials ($t(57) = -3.20$, $p < .01$, $r = .39$). There was no difference in antisaccade error rate between UC and CS trials, ($t(57) = -.03$, *ns*, $r = .00$).

For prosaccade trials, the main effect of preparation time was not significant ($F(1, 57) = 1.57$, *ns*, $r = .16$), but the interaction between trial type and preparation time was significant ($F(1.2, 68.3) = 15.13$, $p < .001$, $r = 43$), reflecting the fact that the difference in errors between the ILT's was greater in the CD trials ($t(57) = -3.30$, $p < .001$, $r = .40$) compared to the UC ($t(57) = 2.16$, $p < .01$ $r = .28$) and CS trials ($t(57) = 2.62$, $p = .01$, $r = .33$ see figure 1.5). For antisaccades participants also made fewer errors when given longer ILT ($F(1, 57) = 45.71$, $p < .001$, $r = .67$). The interaction between trial type and preparation time was not significant for antisaccade trials ($F(2,114) = 1.43$, *ns*, $r = .11$), reflecting the fact that the shorter preparation time increased errors uniformly across trial types (see figure 1.6).

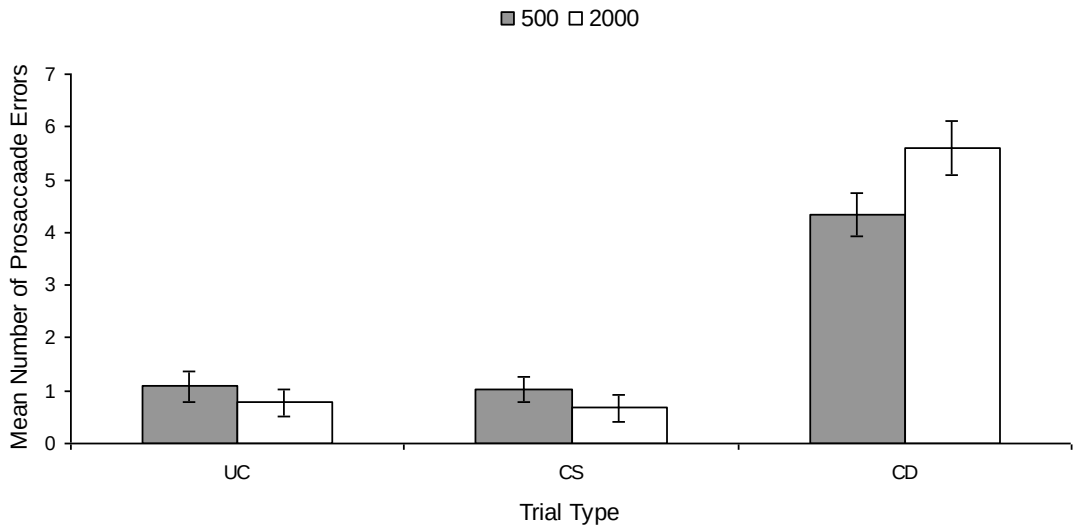

Figure 1.5. Average number of prosaccade errors across trial type, as a function of ILT

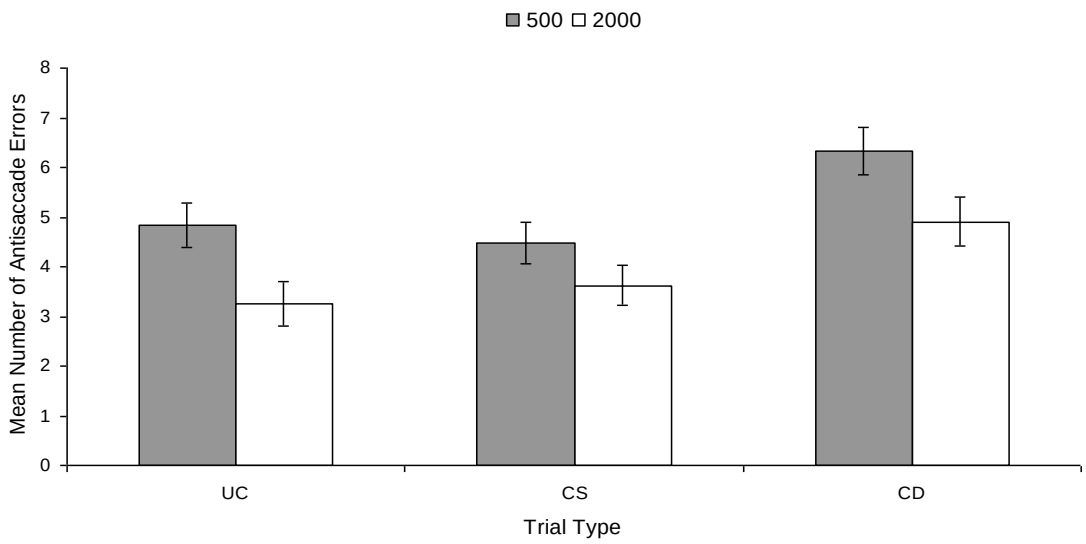

Figure 1.6. Average number of antisaccade errors across trial type, as a function of ILT

Discussion

We explored the effects of cueing on pro and antisaccade performance. Several key findings emerged. With prosaccades, we found that cueing the same location as the target stimulus (CS) resulted in reduced latencies compared to trials that were uncued (UC). Cueing the opposite side (CD) resulted in increased latencies. In addition, participants were faster to make prosaccades when given longer to prepare (2000msec compared to 500msec). Error rate was similar for CS and UC trials, but CD trials increased errors. Preparation time did not significantly alter the number of prosaccade errors (which were very low overall). With antisaccades, compared to UC trials, correct latencies were reduced for CS but not CD trials. Participants were generally faster to make correct antisaccades, and made fewer errors when given longer to prepare. For antisaccade errors, CS trials did not change errors compared to UC trials, but errors were increased in CD trials.

As predicted, participants made more antisaccade errors when the cue appeared on the opposite side to the target (in other words when the cue appeared in the location to which a correct antisaccade should be made). This paradoxical effect replicates Weber et al. (1998). Following Weber et al. (1998) we hypothesized that this effect arises because participants who have successfully adopted the antisaccade ‘task set’ treat the cue as if it were a target stimulus and direct attention to the opposite side

(where the target subsequently appears), thus increasing the likelihood of a capture error. This hypothesis receives some support from the CS latency data, as participants were faster overall on CS trials compared to UC trials. This reduction in correct antisaccade latency on CS trials would be expected if the cue is treated as the target stimulus. If participants have activated the antisaccade task set, the cue would cause attention to shift to the opposite side, which, in the antisaccade task is the correct goal location, thus resulting in a faster subsequent correct antisaccade to that location compared to when no cue is used. However, there was no significant difference in correct antisaccade latencies between CD and UC trials. On CD trials, if participants have activated the antisaccade 'task set', the cue should cause attention to be directed to the opposite side (where the target subsequently appears). On trials where a capture error is avoided, attention needs to be redirected back to the side where the cue had just appeared, which would in turn be expected to result in an increase in correct antisaccade latency compared to uncued trials. Indeed, Weber et al. (1998) found exactly this pattern of results. It is not immediately clear why we did not observe the expected slowing of correct antisaccade latencies in CD trials.

According to the goal activation account, the paradoxical cueing effect found in antisaccade errors should only occur if participants are able to successfully adopt the 'antisaccade set'. We predicted that decreasing the amount of time participants had to prepare for the antisaccade trial would attenuate this effect. In other words, if participants do not have sufficient time to get into the 'antisaccade set', then the cue would be less likely to result in attention being shifted to the opposite side than if the participant was better prepared (e.g. had activated the correct 'antisaccade set' to a higher level). There was evidence that reducing the ILT did result in a significant effect of the amount of time given to prepare for an antisaccade, on both correct latencies and error rate. Therefore, giving participants a short ILT (500msec) to prepare for an antisaccade, significantly increased correct antisaccade latencies and error rate compared to giving participants a longer ILT (2000msec). Although this manipulation worked, we did not observe the predicted interaction between preparation time and cue location for antisaccade errors. In other words the detrimental effect on error rate of cueing the correct goal location was equivalent whether participants had had 500 or 2000msec to adopt the correct antisaccade task set. One possible explanation for the failure to observe the interaction was that the difference between 500 and 2000msec preparation time was not sufficient. In other words, participants had enough time even

in the 500msec condition to sufficiently activate the antisaccade set such that the onset of the cue served to direct attention in the opposite direction. In experiment 2, we tested this hypothesis by reducing the short ILT from 500 to 200msec. The long ILT was reduced from 2000 to 1000msec, as the previous experiment showed that 500msec was sufficient time for participants to adopt an antisaccade set.

Experiment 2

In the second experiment we again explored any potential effects cueing would have on pro and antisaccade performance. Similar to experiment 1, pro and antisaccades were mixed together with cued and uncued trials. The ILTs were shortened to 200 and 1000msec, as 200msec would provide only a very small amount of time for participants to prepare the antisaccade task set. Based on the first experiment, it was predicted that participants prosaccade and antisaccade correct latencies would be significantly shortened in the CS condition, compared to the UC and CD conditions. In addition, we expected to see an interaction between ILT and trial type for antisaccade errors, we expected that the difference in errors between the ILTs would be greater in the CD trials.

Method

Participants

Participants were 30 students from the University of Sussex, 22 female and 8 male, with ages ranging from 19-40, (M = 26.20, SD = 4.36). Undergraduate participants received course credits for taking part and Post graduates volunteered. 15 of the students were given monetary reward, depending on their group. Data from 1 participant was excluded from the final analysis, due to calibration issues.

Apparatus/stimuli

The apparatus and stimuli were identical to experiment 1.

Design/procedures

As in Experiment 1, a within-participants design was used. The mixed pro/antisaccade task contained 192 trials, 96 of which were prosaccades and 96 of which were antisaccades. The procedure was identical to experiment 1, except this time, instruction preparation times were altered to short (200ms), or long (1000ms). Practice trials were given and testing lasted around 20 minutes.

Measures

The measures we used were identical to those used in experiment 1.

Results

Correct prosaccade and antisaccade latencies

A three way 2(task: pro vs. anti) x 3(trial type: UC vs. CS vs. CD) x 2(ILT: 500 vs. 2000) repeated measures analysis of variance, was carried out on prosaccade and antisaccade correct latencies (figure 1.7).

Not surprisingly, the main effect of task was found to be significant ($F(1, 27) = 132.69$, $p < .001$, $r = .91$), as participants correct latencies were faster for prosaccades than antisaccades. A main effect of trial type was also found, ($F(1.73, 46.72) = 43.51$, $p < .001$, $r = .69$, Huynh-Feldt correction, e = .87). Participants made faster saccades in CS trials, than in UC trials ($t(29) = 8.54$, $p = .001$, $r = .85$) but not CD trials ($t(29) = -.60$, $p = .55$, $r = .11$). For prosaccades, participants were significantly faster for CS compared to UC trials ($t(29) = 9.08$, $p < .001$, r = .86). Latencies were also faster for CS compared to CD trials ($t(29) = -9.41$, $p < .001$, $r = .87$). In addition, participants were significantly faster for UC compared to CD trials ($t(29) = -2.65$, $p = .01$, $r = .44$).

For antisaccades: The difference in latencies between UC and CD trials was significant ($t(29) = 3.63$, $p < .001$, $r = .56$), as participants were faster on CD trials. The difference in latencies between CS and CD trials was not significant ($t(29) = -.60$, *ns*, $r = .11$). In addition, a main effect of ILT was found ($F(1, 27) = 18.78$, $p < .001$, $r = .64$), as participants made faster saccades in the long ILT compared to short ILT trials (1000 vs. 200msec).

These main effects were qualified by a significant interaction between trial type and task ($F(1.49, 40.14) = 12.48$, $p < .001$, $r = .49$, Huynh-Feldt correction, e = .74).

The difference in latencies between pro and antisaccades was greater in the CS and UC conditions compared to the CD condition (figure 1.8). The interaction between task and ILT was not significant ($F(1, 27) = .09$, *ns*, $r = .06$), as latencies were faster for both pro and antisaccades, in the long ILT trials compared to short ILT trials. No significant interaction was found between trial type and ILT either ($F(1.63, 43.94) = 1.78$, *ns*, $r = .20$, Huynh-Feldt correction, e = .81), reflecting the fact that latencies on both tasks were always faster for the longer ILT, across all trial types. The three way interaction between task, trial type and ILT was not significant ($F(2, 54) = .39$, *ns*, $r = .08$).

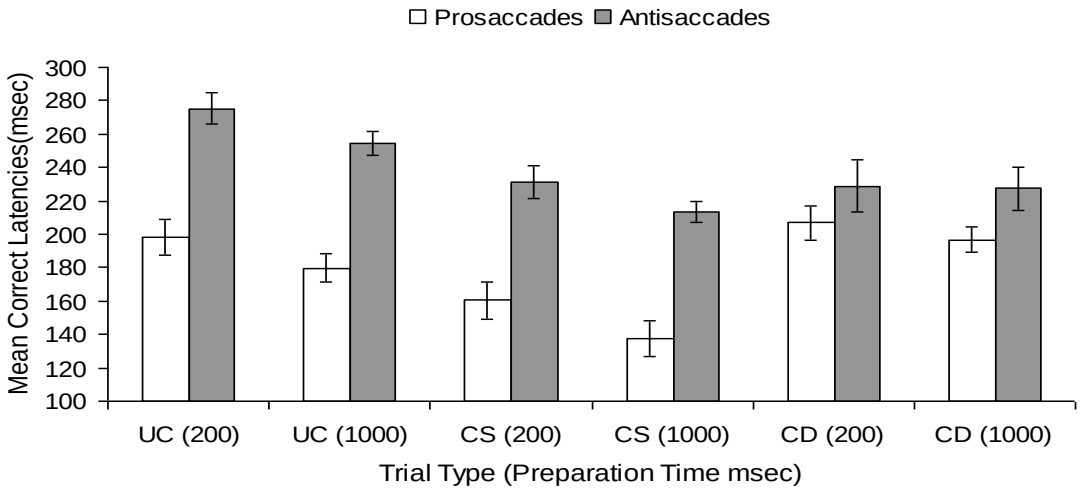

Figure 1.7. Correct saccade latencies across trial type, as a function of task and ILT

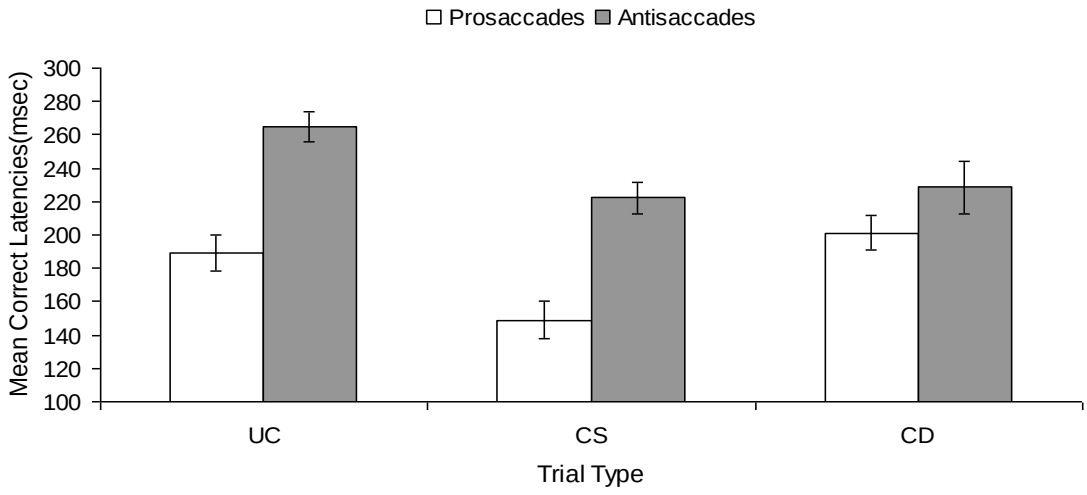

Figure 1.8. Correct saccade latencies across trial type, as a function of task

Prosaccade and antisaccade errors

A three way 2(task: pro vs. anti) x 3(trial type: UC vs. CS vs. CD) x 2(ILT: 500 vs. 2000) repeated measures analysis of variance, was carried out on prosaccade and antisaccade errors (figure 1.9).

A main effect of task was found ($F(1, 29) = 49.43$, $p < .001$, $r = .79$), as more errors were made in the antisaccade task, compared to the prosaccade task. There was also a main effect of trial type, ($F(1.34, 38.78) = 43.90$, $p < .001$, $r = .72$, Huynh-Feldt correction used e = .67). Errors were greater in the CD condition, compared to CS ($t(29) = -3.94$, $p < .001$, $r = .59$) and UC conditions ($t(29) = -3.92$, $p = .001$, $r = .59$). There was no difference between CS and UC trials ($t(29) = -.05$, $p = .96$, $r = .01$). In addition, a main effect of ILT was also found, as participants made more errors when given less (200msec) preparation time ($F(1, 29) = 16.03$, $p < .001$, $r = .60$).

These main effects were qualified by some interactions. Firstly, there was a significant interaction between task and ILT ($F(1, 29) = 7.92$, $p < .01$, $r = .46$), because for prosaccades, error rate was similar for both short (200msec) and long (1000msec) ILT, whereas for antisaccades, errors were reduced with longer preparation time (figure 1.10). A significant interaction also arose between trial type and ILT ($F(2, 58) = 7.72$, $p = .001$, $r = .34$) as participants made fewer errors with longer preparation time for UC and CS trials, but slightly more errors with longer preparation time for CD trials (see figure 1.11).

There was no significant interaction between trial type and task ($F(1.42, 41.29) = .29$, *ns*, $r = .08$, Huynh Feldt, e = .71), as the difference in errors between pro and antisaccades was the same for all trial types. The three way interaction between task, trial type and ILT was not significant ($F(1.67, 48.56) = .74$, *ns*, $r = .12$, Huynh-Feldt, $e = .84$).

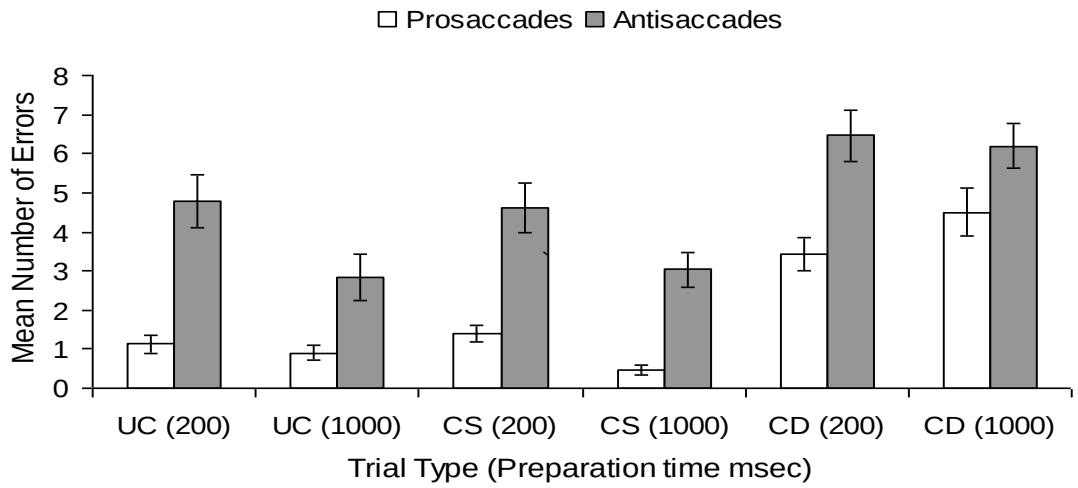

Figure 1.9. Average number of pro and antisaccade errors across trial type as a function of task and ILT

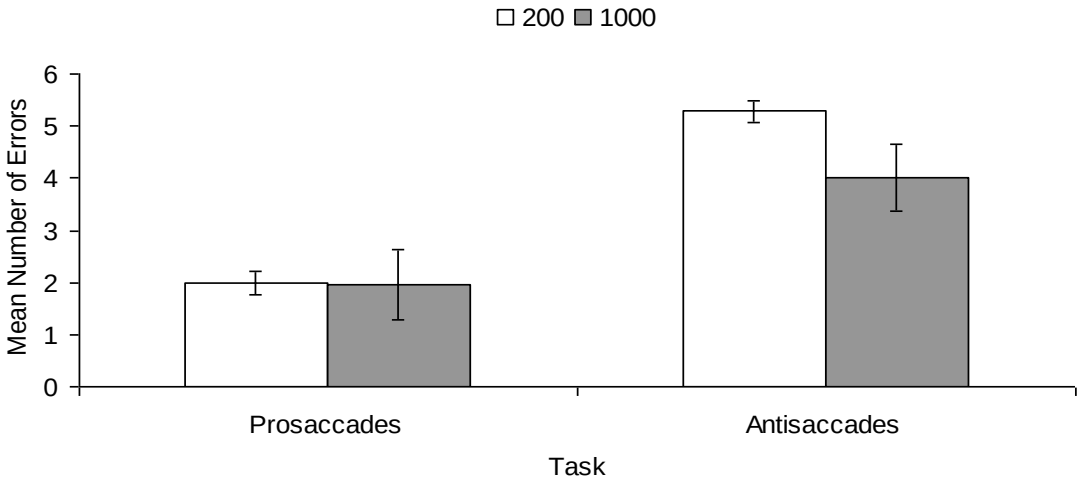

Figure 1.10. Average number of pro and antisaccade errors across task as a function of ILT

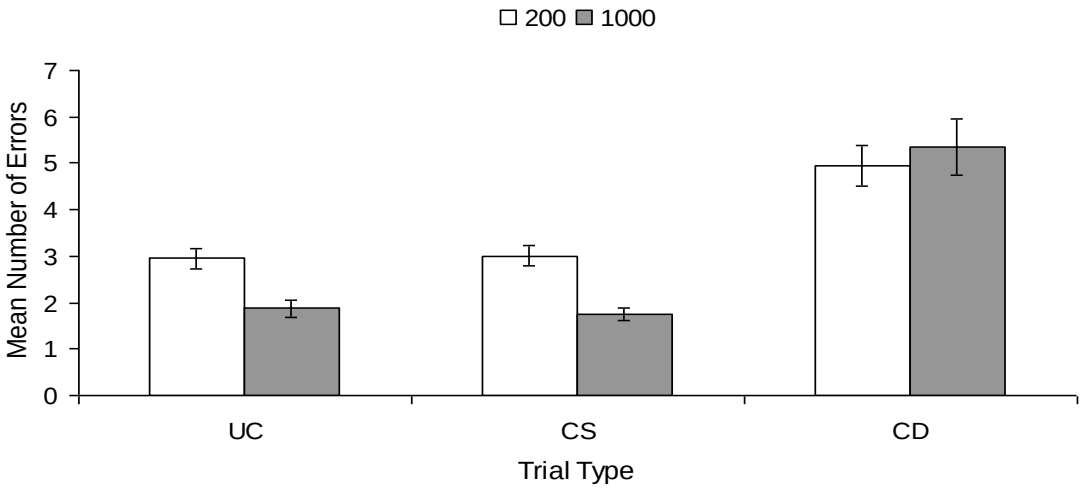

Figure 1.11. Average number of pro and antisaccade errors across trial type as a function of ILT

Discussion

In the second experiment, we again explored the effects of cueing on pro and antisaccade performance. All current prosaccade findings replicated the prosaccade findings from the first experiment. We found reduced correct latencies, when trials were cued the same side as the target stimulus (CS), compared to trials that were uncued (UC). Participants were faster to make prosaccades when given longer to prepare (1000 vs. 200msec). Error rate was similar for CS and UC trials, but CD trials increased errors. For antisaccades, compared to UC trials, latencies were reduced for CS and CD trials. For antisaccade errors, participants made a similar amount of errors on CS and UC trials, but CD trials increased errors. Longer preparation time resulted in less antisaccade errors, compared to shorter preparation time and participants were generally faster to make antisaccades when given longer to prepare.

Cueing the opposite side to the target stimulus (i.e. goal location) has again paradoxically increased antisaccade errors. This supports the suggestion that the cue serves as a go signal in participants who have sufficiently activated the antisaccade task set, thus diverting attention away from the cue, towards the location of the upcoming target. This increases the likelihood of participants making an antisaccade error. We predicted that decreasing the amount of time participants had to prepare for an antisaccade trial would attenuate the extent to which they could successfully activate the

antisaccade ‘task set’, making them less likely to mistakenly direct their attention to the opposite side of the cue, as opposed to the target, and therefore less vulnerable to cueing effects. In support of the first study, participants were generally slower and made more errors when given shorter ILT (200msec) compared to longer (1000msec). Despite reducing the shortest ILT to only 200msec, the expected interaction between antisaccade ILT and trial type was not found. Again, the detrimental effect on error rate of cueing the correct goal location was equivalent whether participants had 200 or 1000msec to adopt the correct ‘antisaccade task set’. This finding suggests that either 200msec was a sufficient amount of time to activate the ‘antisaccade set’, or that reducing the amount of preparation time given to make an antisaccade has no influence on the paradoxical cueing effect. Problems with the hypothesis arise, as it seems the amount of ILT is not entirely relevant in terms of altering the cueing effect. However, as the ILT manipulation was successful, this suggests that ‘task set’ is a useful concept.

Combined analysis

In order to be certain that the separate analyses had not missed ILTs potential impact on the cueing effect, or those previous null results were not due to a lack of power, we carried out a combined analysis, using the total N of 88. We used CS and CD antisaccade error rates from the smaller ILTs (exp 1 = 500msec, exp 2 = 200msec) and compared them to CS and CD antisaccade error rates from the longer ILTs (exp 1 = 2000msec, exp 2 = 1000msec). UC error rates were not included, as we were only interested to see if the difference in error rate was greater for CD trials.

A two way 2(trial type: CS vs. CD) x 2(ILT length: short vs. long) repeated measures analysis of variance, was carried out on antisaccade errors.

The interaction between trial type and length of ILT was not significant, ($F(1, 87) = .03$, *ns*, $r = .02$), as the shorter length ILT did not result in a bigger difference between errors for CS and CD trials (figure 1.12). This finding suggests that reducing the amount of time participants had to prepare for an antisaccade trial, did attenuate the ‘antisaccade set’ but did not have the predicted impact on the cueing effect. It would appear that 500 and 200msec ILTs were still sufficient times for participants to activate the antisaccade ‘task set’.

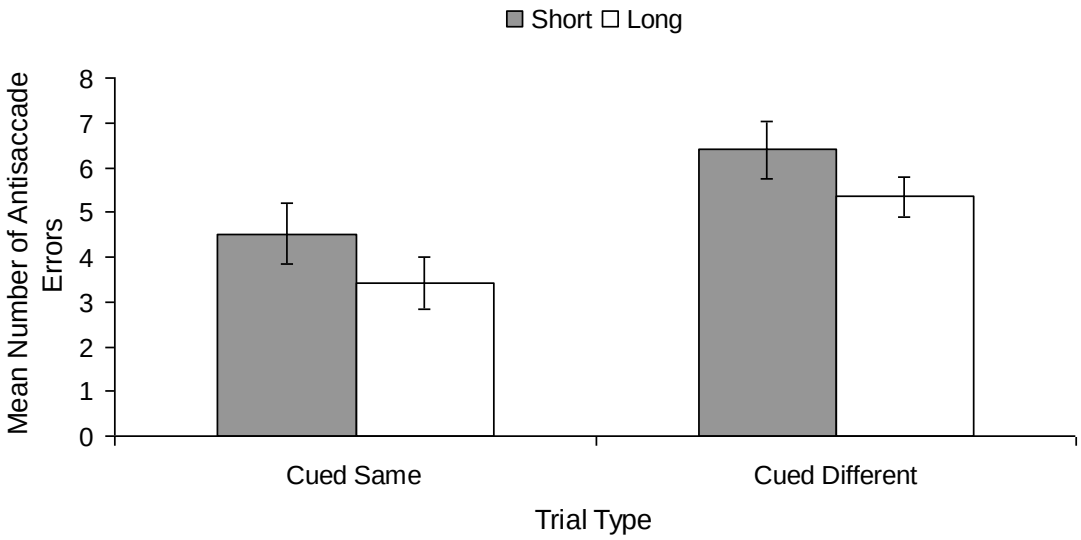

Figure 1.12. Average number of antisaccade errors across trial type as a function of ILT length

According to the goal activation account of the paradoxical cueing effect, the effect should be greatest in those participants who are most successful in adopting the 'antisaccade task set' – in other words those participants who make fewest errors. In order to test this hypothesis we calculated the magnitude of the cueing effect for each participant to determine the size of the cueing effect, by subtracting the number of CS errors from CD errors. By doing this, we were able to see if those who make significantly more CD errors than CS errors were actually those who make fewest UC errors.

To explore the relationship between magnitude and antisaccade uncued error rate, we ran a correlational analysis, with the prediction that a high magnitude would mean fewer uncued errors and a low or negative magnitude would mean more uncued errors. A significant negative correlation arose between uncued errors and magnitude (r = -.32, $p < .01$), as those who make fewest errors on UC trials, are those who have the biggest magnitude, i.e. they are the people whose performance gets 'hit' by cueing the different side the most (figure 1.13).

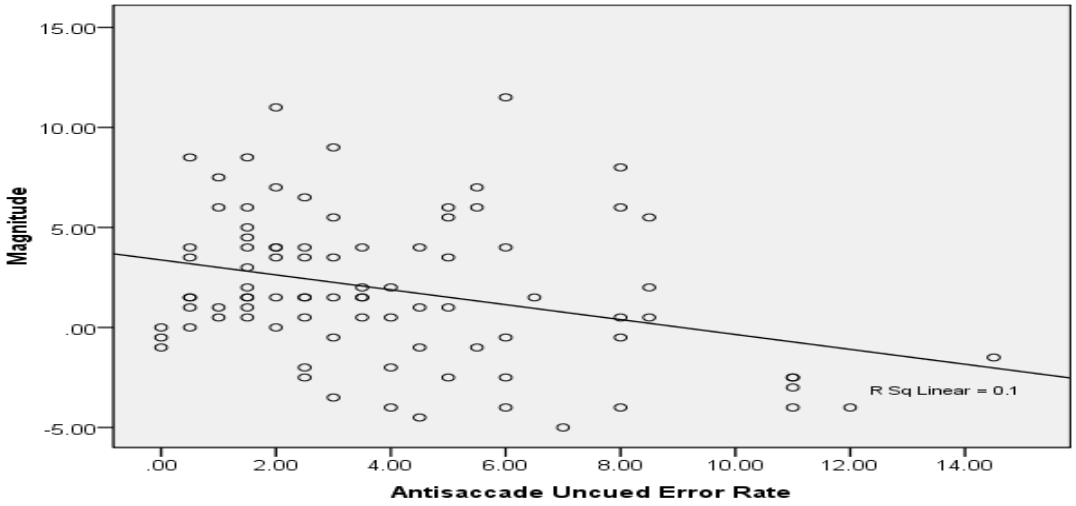

Figure 1.13. Relationship between antisaccade error rate and magnitudes

General Discussion

Across two experiments, we investigated the effects of transient cues on pro and antisaccade performance. In both studies, cueing the correct goal location for antisaccades (CD) had the paradoxical effect of increasing antisaccade error rate, whereas cueing the same side as the target (CS) had no impact on errors. Correct antisaccade latencies were reduced with CS trials. However, CD trials also decreased correct latencies. Despite attempts to inhibit the 'antisaccade set', reducing instruction lead time (ILT) did not reduce the cueing effect. Using peripheral cues had greater impact on antisaccade performance than prosaccade performance. In terms of prosaccades, CS trials decreased correct latencies, whereas CD trials increased latencies, compared to trials that were uncued (UC).

The finding that cueing the location to which a correct antisaccade should be made actually increases the number of antisaccade errors supports previous work (Fischer & Weber 1996; Weber *et al.*, 1998) and suggests that participants treat the cue as if it were the target and direct attention to the opposite side (where the target subsequently appears – thus increasing the likelihood of a capture error). Even though participants were cued to where they should saccade to, on the whole attention was captured sufficiently for erroneous prosaccade errors to occur. The relationship between magnitude and UC errors confirmed that those who made the most CD compared to CS errors, were those who made the least UC errors. This finding supports the suggestion that the paradoxical effect of increasing errors when the correct location is cued occurs because

participants have successfully adopted the antisaccade 'task set'. Those who are most successful at activating the 'antisaccade set' (e.g. those who make fewest errors in the uncued trials) are more likely to make CD errors, as they treat the cue as a go signal and shift attention to the location in which the target subsequently appears.

The present results showed a decrease in correct antisaccade latencies when antisaccade trials were cued to the goal location (CD), compared to uncued trials (UC). This finding does not support Weber et al. (1998) who found latencies were increased when the cue occurred in a different location to the target stimulus. They interpreted this increase in latency in CD trials as an allocation of attention at the opposite side that is elicited by an automatic orienting mechanism cue. Consequently, a subsequent orienting to the correct position (opposite to target) is necessary before the correct antisaccade can be made, all of which adds to the reaction times of the correct antisaccades. According to the goal activation hypothesis (Nieuwenhuis et al., 2004), if the 'antisaccade set' is sufficiently activated, then cueing the opposite side to the target should increase correct antisaccade latencies (as found by Weber et al., 1998). If the cue serves to direct attention to the opposite side, (to the location in which the target subsequently appears, and thus the incorrect location for an antisaccade) attention would need to be redirected back to the side that was cued, thus resulting (on those trials in which a capture does not occur) in an increase in correct antisaccade latencies compared to uncued trials (in which attention simply has to be directed to the goal location). It is not clear why, in our results, we failed to replicate this finding. In experiment 1 there was no difference, between UC and CD latencies. In experiment 2 we actually observed a significant decrease in correct antisaccade latencies when the goal location was cued compared to uncued trials. One speculative explanation might be that in the mixed task we employed, the cue acts as a general go signal, which commands the preparation of any oculomotor movement, much faster than if there was no cue at all. In other words, CD trials contain additional information (cue) that can be used to shorten the time needed to saccade to the goal location.

In experiment 1, we observed a decrease in errors, but no difference in correct antisaccade latencies in CD compared to UC trials. In experiment 2 we observed both decreased correct antisaccade latencies and increased antisaccade errors on CD compared to UC trials. According to parallel programming accounts or race models of antisaccade performance (Massen 2004; Munoz & Everling, 2004; Reuter & Kathmann, 2004; Reuter et al., 2005), at target onset, a race begins between activity in the separate

prosaccade and antisaccade pathways, with the winner reaching the threshold for triggering a saccade first. According to these accounts, any manipulation that differentially affects pro and antisaccade latencies will consequently influence antisaccade error rate, as it will influence the likelihood of one of the pathways reaching threshold first. According to Massen (2004), a manipulation that results in significantly increased antisaccade correct latencies should either result in an increase in antisaccade error rate or no change in error rate if the manipulation also increases prosaccade latencies to a similar degree. By the same token, a manipulation that decreases antisaccade correct latencies should result in a decrease in error rate providing it does not also decrease prosaccade latencies to the same extent. In experiment 2, CD trials reduced correct antisaccade latencies (whilst increasing prosaccade latencies) compared to UC trials, but we did not observe a decrease in antisaccade errors (in fact we found an increase) for CD trials, as would be predicted by parallel programming models. One potential explanation as to why our finding (reduction in latencies resulting in an increase in error rate) is not in line with parallel programming account predictions (i.e. reduced latencies resulting in reduced errors), is that perhaps parallel programming accounts are more applicable to the time course of standard antisaccade trials (uncued trials). The race model assumes that activity in the separate pro and antisaccade pathways begin the race for saccade execution at target onset, but any manipulation before or at target onset (such as a peripheral cue), can facilitate attention and consequently affect the outcome of the race. Therefore, presenting a visual cue before target onset could mean the race between the separate prosaccade and antisaccade pathways was already activated, and this additional visual information (e.g. cues) may had provided an advantage to the prosaccade pathway, resulting in an erroneous prosaccade reaching the threshold for triggering a saccade before a correct antisaccade.

In experiment 1, we did not observe the predicted interaction between trial type and instruction lead time (ILT). It was predicted that the shorter ILT (500msec), would reduce the amount of time for participants to activate the ‘antisaccade set’, thus reducing the likelihood that the cue would be treated as a go signal and result in a shift of attention to the opposite side. However, this pattern of results did not arise. One potential explanation of this was that the difference between 500 and 2000msec preparation time was not sufficient. In other words participants had enough time even in the 500msec condition to sufficiently activate the ‘antisaccade set’. This explanation does not however appear to be sufficient, as in experiment 2 we changed the preparation time

difference to 200 and 1000msec and still did not observe a reduced cueing effect for the shorter ILT (200msec). It would appear that participants had enough time to correctly activate the 'antisaccade set' even when given 200msec to prepare.

However, in both experiments, the shorter ILTs (exp 1 500msec, exp 2 200msec) increased antisaccade errors compared to the longer ILTs (exp 1 2000msec, exp 2 1000msec), across all trial types. This would suggest that the longer you have to prepare for the concurrent pro or antisaccade, the more likely it is an error will be avoided, regardless of whether a cue appears or not. Furthermore, closer inspection of the shorter ILTs, shows almost exactly the same error rate (500msec = 5.22 vs. 200msec = 5.28). This implied that although reducing ILT impacted on antisaccade error rate and could therefore be argued to have influenced how effectively participants were able to activate the 'antisaccade set', it did not result in any amelioration of the paradoxical cueing effect. In addition, by subtracting CS errors from CD errors, we were able to determine the nature of the cueing effect. This enabled us to see if those who made more CD errors compared to CS errors, would be those likely to make fewest UC errors. As previously mentioned, we found a relationship between magnitude and UC error rate. Participants, who had a higher positive magnitude, were those who were affected more by CD trials, meaning they made less UC errors. In contrast, those who had a negative magnitude were those who were not affected by the cue as much as they made more errors on UC trials. This correlation supports the fact that those who are better able to activate the 'antisaccade set' will succumb to more antisaccade errors when the cue is different from the target. The correlation between uncued antisaccade errors and magnitude supports the idea that 'task set' activation is important for antisaccade performance, but does not seem entirely relevant to the cueing effect, as the interaction between trial type and ILT was not observed. One possible explanation as to why this interaction was not found is that perhaps as long as the 'task set' is slightly activated, then this will result in an increase to antisaccade errors for CD compared to CS trials.

Across two experiments, we observed an increase in antisaccade errors when a peripheral cue was actually cueing the correct goal location, compared to when the incorrect side was cued. Although paradoxical, this 'cueing effect' should be reduced if participants have less time to activate the antisaccade 'task set', but this was not the case. This suggests that as long as the 'task set' is minimally active, then the cueing effect will occur. However, the 'task set' is a useful concept and highly applicable to the antisaccade task, because latencies and errors were affected by the amount of

preparation time given. The current findings suggest that those who are better able to activate the ‘antisaccade set’ will be more vulnerable to the cueing effect, resulting in a higher magnitude score and fewer UC errors, than those who are less able to activate it. Taken together, the present findings suggest participants’ are using visual cues as part of their saccadic programming, to extract information about the direction and location of the next saccade.

Article 2 - Incentive and Antisaccade Performance: A Rewarding Influence

Abstract

Correct antisaccade performance requires participants to refrain from looking at a sudden onset target, and instead initiate a saccade to its mirror image location. The task thus provides researchers with a convenient tool to investigate the relative influence of top down and bottom up processes on behaviour. Previous research has shown that incentives (typically monetary) can improve performance on a range of cognitive tasks, but the effects of incentives on antisaccade performance are not clear. The small number of studies addressing this issue have produced inconsistent results. It is also not clear whether non-financial incentives can influence antisaccade performance. In our first experiment, participants performed the antisaccade task with either no incentive, a verbal incentive, or a financial incentive. Neither incentives significantly reduced antisaccade error rate, but correct latencies were significantly reduced in the financial incentive condition. In a follow-up experiment, we sought to disentangle the effects of trial by trial feedback and financial incentive on antisaccade performance. Compared to the no incentive condition, correct antisaccade latencies were significantly reduced by both trial by trial feedback (in the absence of financial incentive) and a financial incentive (in the absence of trial by trial feedback). In a final experiment, we used a within-participants design to try to elicit an effect of incentive on antisaccade errors and compared a condition in which financial incentive and feedback were given with a condition in which financial incentive alone was given. Errors were reduced in the combined incentive + feedback condition compared to the no incentive condition. Participants were faster to make a correct antisaccade when given financial incentive alone, but somewhat surprisingly this was not the case for the combined feedback and incentive condition. Together these findings confirm that antisaccade performance can be improved by financial incentives, and demonstrate that trial by trial feedback can also result in performance improvements, suggesting that awareness of errors helps participants to monitor and adjust ongoing task performance.

Introduction

The antisaccade task, developed originally by Hallet (1978), requires participants to refrain from making a prosaccade towards a sudden onset target and instead make an eye movement in the opposite direction, typically to an equidistant position in the opposite hemifield. It is this competition of an exogenously driven, prepotent response, with an endogenously driven volitional one that has led to the adoption of the antisaccade task by psychologists and cognitive neuroscientists interested in studying goal directed behaviour.

Healthy participants typically fail to suppress erroneous prosaccades towards the target on around 20–25% of trials. These "antisaccade errors" are quickly corrected with a saccade towards the mirror image location (e.g. Fischer and Weber 1992; Smyrnis et al., 2002). Patients with damage to the dorsolateral prefrontal cortex (Pierrot-Deseilligny et al. 2005) and patients with schizophrenia (Hutton et al., 1998) both have increased error rates compared to healthy controls.

According to current models of antisaccade performance, at target onset, a "race" ensues between activity in the neural system supporting the prosaccade towards the target, and activity in the neural system supporting the correct antisaccade response. The "winner" is the system whose activation reaches the threshold for saccade triggering first (Massen 2004; Munoz & Everling, 2004; Reuter & Kathmann, 2004). These "parallel programming" or "accumulator" models predict that the faster a correct antisaccade can be programmed the more likely it is to win the race, and therefore the less likely an error is to occur.

Competitive race models of antisaccade performance are supported by converging evidence. Behavioural studies in healthy populations have shown that experimental manipulations which result in increased antisaccade, but not prosaccade latency, also result in an increase in antisaccade errors (e.g. Massen, 2004). In addition, antisaccade error rate is strongly correlated with prosaccade latency in healthy populations (Roberts, Hager & Heron, 1994; Taylor & Hutton, 2007). Patient populations that demonstrate increased antisaccade error rates also have prolonged correct antisaccade latencies (Guitton, Buchtel & Douglas, 1985; Hutton, Joyce, Barnes & Kennard, 2002). Neurophysiological research in non-human primates has found that the frontal eye fields (FEF) and the superior colliculus (SC) play an important role in

the antisaccade task (Everling, Dorris & Munoz, 1998; Everling & Munoz, 2000; Sato & Schall, 2003). Particularly, firing patterns of saccade neurons in the FEF and SC are believed to account for the variability in saccadic reaction times (Hanes & Schall, 1996). An important finding is that antisaccade neuron activity is generally lower in the FEF before an antisaccade error compared to before a correct antisaccade (Munoz & Everling, 2004). Finally, Neuroimaging research, using electroencephalography (EEG), has found that antisaccade errors are associated with a reduction in the amplitude of the presaccadic negativity, an event related potential (ERP) typically observed 60msec before saccades (Evdokimidis, Mergner, & Lucking, 1992).

Another feature of competitive race models, is that they can accommodate the extensive evidence suggesting that antisaccade performance is modulated by both endogenous "top down" processes (e.g. those involved in maintaining the intention to perform an antisaccade) and exogenous "bottom up" factors (e.g. size and spatial position of the sudden onset target). The amount of time activity in the neural systems supporting the antisaccade takes to reach threshold could reflect baseline levels of activity prior to target onset, as well as the subsequent rate of rise. Experimental manipulations that alter baseline levels of activity in the neurons supporting the antisaccade would be expected to increase correct antisaccade latency, and consequently increase error rate. Massen (2004) observed this pattern of results by manipulating the frequency of antisaccade trials within blocks. In blocks where antisaccade trials were comparatively rare, correct antisaccade latencies and antisaccade errors were increased compared to blocks in which antisaccade trials dominated. One interpretation of this finding is that baseline levels of activity in the antisaccade system were lower in the low frequency blocks compared to the high frequency blocks.

Another way in which top down influences on behaviour can be moderated is through the use of rewards. Within the research literature, rewards are generally divided into two main categories, primary rewards and secondary rewards. Primary rewards comprise food, water, shelter and sex. Secondary rewards include security, praise, pleasant touch, music and money. Typically, non-human primate research has used food/water as an incentive, (e.g. Kawagoe, Takikawa, & Hikosaka, 1998; Watanabe, Lauwereyns, & Hikosaka, 2003) whereas human research generally uses financial incentives (e.g. Elliot, Newman Longe, & Deakin, 2003; Knutson, Westdorp, Kaiser, & Hommer, 2000).

Financial incentives have been found to improve performance on a range of cognitive tasks. For example, Heitz, Schrock, Payne, & Engle (2008) investigated the effects of incentive on the reading span task (Daneman & Carpenter, 1980) in high and low working memory span participants. Heitz et al. (2008) used an adapted version of the task where participants had to read sentences aloud and recall a letter that was presented after each sentence at a later time. Participants were either given no incentive, a feedback incentive (visual feedback stating how many correct letters were recalled), or feedback and monetary reward (visual information about how much each trial was worth and how much the subtotal was). They found that participants who received feedback and monetary reward combined recalled significantly more letters (increased reading span) compared to those who received just feedback or no incentive. This result suggests that incentive is able to improve reading span performance in participants with high and low working memory ability.

A number of studies have explored the relationship between saccadic eye movement tasks and rewards. For example Kawagoe et al. (1998) investigated the relationship between saccadic eye movement and rewards in monkeys. They trained two monkeys to perform a memory-guided saccade task whereby they had to saccade to the location of a previously remembered target stimulus. Single unit recording was carried out and eye movements were recorded using a search coil method. A correct response (saccade to the location of the previously shown target) was only rewarded for saccading to 1 of 4 possible goal locations. The reward was water. The authors found that reward expectancy modulated memory-related and visual responses of projection neurons (neurons with low spontaneous activity) in the caudate nucleus (an area associated with motivation, Robbins & Everitt, 1996). Caudate neuron activity depended on whether a correct trial would be rewarded immediately or not, therefore activity was reduced when the reward was not expected immediately and activity was increased when a reward was expected immediately. Saccade velocity and saccade latencies were related to the modulation of caudate neurons by reward. When caudate neuron activity was enhanced (reward expected) and the reward followed the saccade, the latency of the saccade to the location of the previously shown target was reduced. In other words, correct latencies were faster when incentive was given. At the same time, this increase in activity resulted in an increase in saccade velocity. These findings suggest that the caudate links visual information with motivational information in order to help initiate the programming of saccades.

In a more recent study using electrophysiological recordings, Watanabe, Lauwereyns, & Hikosaka (2003) trained monkeys to perform prosaccade trials, whereby a saccade to the target signified a correct response. They wanted to explore the effects of motivational context on saccade-related activity in caudate neurons, as previous research has shown that the caudate is involved in both oculomotor initiation and motivational information in non-human primates (Kawagoe et al., 1998). An auditory feedback signal was sounded after each completed trial, and a drop of water was given after half of the completed trials. In another block, reward was mapped onto one target position only. They found a correlation between activity in the caudate nucleus and rewarded and unrewarded eye movements. Specifically, caudate neuron activity facilitated saccadic programming by reducing the latency of the saccade to the target on both rewarded and unrewarded trials. This suggests that the monkeys were motivated enough to make prosaccades, even if they were not rewarded for each correct response. Correct prosaccade latencies were faster for rewarded trials compared to unrewarded trials. Taken together, these findings suggest a strong link between reward and saccadic eye movement in the caudate nucleus as the expectation of receiving a reward can modulate the latencies of saccades to a target and to a previously remembered target location. One aim of the current research was to see if this relationship between saccadic eye movement and reward would extend to antisaccades.

A number of researchers have investigated the effect of incentive on antisaccade performance. For example, Amador, Schlag-Rey, & Schlag (2000) measured cortical activity in monkeys performing rewarded antisaccade trials. On correct trials, apple juice was given and a flash of light was shown to the monkey, but no light flash or juice was given on incorrect trials. They identified two types of reward-related neurons in the supplementary eye fields (SEF) that were distinguished by their patterns of mutual firing - reward predicting and reward detecting. Reward detecting neurons fired in line with the delivery of the reward and reward predicting neurons fired prior to saccade onset. They concluded that the reward predicting and reward detecting neurons may act as an aid for learning the reward task. However, as the authors have not reported any latency data, it is difficult to infer the effects of incentive on antisaccade trials in non-human primates.

Duka & Lupp (1997) were the first to investigate the effects of incentives on the antisaccade task in humans. They gave participants separate blocks of prosaccades, memory-guided saccades and antisaccades and predicted that incentive would improve

accuracy on memory-guided and antisaccades only. The high working memory demands of the antisaccade task mean a degree of motivation is required to make a correct antisaccade and the authors reasoned that incentive could modulate this motivation by improving antisaccade accuracy. Antisaccade trials with incentive were performed separately to trials with no incentive. On blocks containing incentive trials, participants were informed that they would be given an honorarium (50 Deutsch Marks) for particularly good task performance. Furthermore, participants were told they had to exceed the best score of performance they reached during practice. The authors did not find any effect of incentive on the number of correct prosaccades made, or on correct prosaccade latencies. However, they did observe an effect of incentive on antisaccade accuracy, as the number of correct antisaccades was increased compared to the no incentive condition. They did not find any effect of incentive on correct antisaccade latencies. Their findings suggest firstly, that simply telling participants at the beginning of the study they will be rewarded money for good performance was enough to enhance antisaccade performance. The results also suggest that antisaccade accuracy is more sensitive to an incentive manipulation than correct antisaccade latency. Critically, the authors do not report the number of antisaccade trials completed by each participant; therefore it is difficult to know if the data is reliable.

Recently, other researchers have looked at the effects of incentives on antisaccade performance. Blaukopf & Di Girolamo (2005 exp. 2) used a visual cueing paradigm in which they assigned a motivational value to the go-signal on each trial (blue = 10p, pink = 1p). They also asked participants to press a button on the keypad if they thought they made an antisaccade error. Interestingly, they found the value of the go-signal had no impact on the amount of antisaccade errors, or on correct latencies. However, compared to low reward trials (1p) error latencies were faster in high reward trials (10p), but only on trials in which the participants recognized that they had made an error (participants are typically unaware of around 50% of their errors, Mokler & Fischer, 1999). The authors suggested that the difference in error latencies between high and low reward trials occurred because before an antisaccade error is made, participants are able to extract information about the go-signal (i.e. its value and location). It is possible the authors did not find an effect of incentive on error rate or correct latency because the magnitude of the reward was not large enough to motivate participants to improve their performance.

In a follow up study, Blaukopf & Di Girolamo (2006) investigated the influences of both reward and punishment on antisaccade performance. Again, the go-signal indicated how much money could be won or lost on an individual trial (blue, pink, green or orange) and participants could win or lose 1p or 25p per trial. At each rest period (after 72 trials) participants were told how much their subtotal earnings were. There was no effect of incentive on antisaccade errors. Contrary to their previous study, they found that correct latencies were slower in high-reward trials (25p) compared to low-reward trials (1p). Their findings do not support those of Duka & Lupp (1997), who found no effect of incentives on correct antisaccade latency. This discrepancy may have occurred due to some methodological weaknesses in Balukopf & Di Girolamo's work. Firstly, unlike the Duka & Lupp study, in which antisaccade performance was compared in blocks of incentive or no incentive trials, in Blaukopf & Di Girolamo's study, it was the go-signal that indicated how much could be won or lost on each trial. The go-signal was only present for 650msec and it is possible that participants did not have enough time to adequately process the valence (win or lose 1p or 25p) of the trial before the target appeared and a response had to be made. Secondly, participants had to remember the four colour/financial outcome rules throughout the task. Increased processing load could have impacted on antisaccade performance. Finally, the authors did not incorporate any neutral trials, where no reward or punishment occurs. Neutral trials would have enabled them to have a baseline measure of antisaccade performance, which is important for making comparisons between standard antisaccade trials and incentive antisaccade trials.

Other studies have also investigated the effect of monetary reward on antisaccade performance. In a study by Jazbec, McClure, Hardin, Pine, & Ernst (2005), clinically anxious, depressed or healthy adolescents made prosaccades and antisaccades where either a correct trial was rewarded, an incorrect trial was punished, or there was no reward or punishment. As with Blaukopf & Di Girolamo's studies, the initial cue indicated on each trial whether a prosaccade or antisaccade had to be made and also indicated the valence of the trial (+ = reward, - = punishment, O = neutral). Participants could win or lose $1 on reward and punishment trials respectively and feedback on how much was won or lost was given after each trial. Healthy participants showed reduced antisaccade error rates on reward trials compared to neutral trials, but correct antisaccade latencies were not reduced when reward was given. Their findings support Duka & Lupp (1997) as error rates were reduced when incentive was given, suggesting

the magnitude of the reward was enough to modulate antisaccade error rate. However, this finding does not support Blaukopf & Di Girolamo (2006) who did not find an effect of incentive on antisaccade errors, suggesting that differences in experimental design may be responsible for this discrepancy. The authors did not report prosaccade results. It is possible that Jazbec et al. (2005) did not find an effect on correct antisaccade latencies because antisaccade trials were interleaved with prosaccade trials, thus a true measure of antisaccade performance (i.e. a pure antisaccade block) was not taken. Also, it is difficult to draw firm conclusions about these findings as the authors did not use adults and adolescents have increased error rates compared to adults (Klein & Foerster, 2001).

In another study, Jazbec et al. (2006) used healthy adolescents and healthy adults to look at the effects of reward and punishment on mixed prosaccade and antisaccade performance. The design was identical to their previous study. Both adolescents and adults made more correct antisaccades when given reward, and replicating their previous finding, incentive did not alter correct antisaccade latencies.

One issue with studies that have explored the effects of incentive on antisaccade performance is that if the delivery (or withholding) of the incentive occurs on a trial by trial basis, participants receive feedback concerning their performance. Providing feedback, after each trial or at regular intervals, has the potential to motivate task performance, regardless of whether a financial incentive is given as well. Feedback might be a particularly powerful moderator of antisaccade performance, because research has found that participants are generally unaware of a large portion of antisaccade errors (Mokler & Fischer, 1999). Recent cognitive control models (Botvinick, Braver, Barch, Carter, & Cohen, 2001; Miller & Cohen, 2001) state that the anterior cingulate cortex (ACC) and the dorsolateral prefrontal cortex (DLPFC) comprise a closed feedback circuit; when conflict is detected, the ACC will signal for increased cognitive control from the DLPFC. These models suggest that post error slowing may occur if the previous trial was erroneous and this slowing is a result of increased cognitive control, to increase the chance of a correct response. In other words, knowledge of correct trials can be used to adjust performance on future trials. It could be argued that effects observed by Jazbec et al. (2005, 2006) may have been due to trial by trial feedback as opposed to financial incentive. One aim of the present research was to look at the combined effects and separate effects of monetary incentive and feedback, to see if one confounds the other.

In a third study, Hardin, Schroth, Pine, & Ernst, (2007) again explored the relationship between incentive and antisaccade performance, in groups of anxious, depressed, healthy adolescents and a group of healthy adults. In this experiment, only antisaccades were required and a saliency manipulation was added, both rewards and punishments could be low, medium or high in value ($1, $2, $4). One impact of this manipulation was participants could win more money than in their previous studies. Participants completed four blocks of 56 antisaccade trials and participants were given trial by trial feedback of their earnings. Antisaccade errors were reduced in reward trials compared to neutral trials in healthy adults consistent to their previous study. In addition to reducing error rate, this study found reward trials reduced correct antisaccade latencies compared to neutral trials. This latter finding contradicted their earlier studies (Jazbec et al., 2005, 2006) and may have occurred because they changed the design from mixed pro/antisaccades to pure antisaccades. It is therefore possible that the antisaccade-only design is more sensitive to correct antisaccade latencies than the mixed design.

Together, these studies do not paint a consistent picture of the effects of incentive on antisaccade performance. Perhaps the most consistent finding is that providing a monetary reward can reduce antisaccade error rate in healthy adults (Duka & Lupp, 1997; Hardin et al., 2007; Jazbec et al., 2006, but see (Blaukopf & Di Girolamo, 2005, 2006). One study found that financial incentive reduced correct antisaccade latencies (Hardin et al., 2007; but see (Blaukopf & Di Girolamo 2005, 2006; Duka & Lupp 1997; Jazbec et al., 2005, 2006). It is likely that the large differences in task parameters and experimental design between studies are a contributing factor to these inconsistencies.

The experiments reported here aim to address a number of methodological weaknesses identified in the previous research, such as mixing antisaccades with prosaccades, or having only two goal locations. Although most of the studies have included a feedback element to their designs, they have not taken into consideration the fact that providing feedback may actually be able to modulate participants' antisaccade performance in its own right. Consequently, the present research incorporated alternative incentives to money, including verbal incentive and trial by trial feedback, to see if they were able to reduce antisaccade errors or correct antisaccade latencies, or both. Finally, we sought to extend previous research by establishing whether non-financial incentives are capable of modulating antisaccade performance.

In the first experiment we compared antisaccade performance in three conditions, no incentive, verbal incentive, financial incentive. In the no incentive condition, participants simply completed a block of standard antisaccades. In the verbal incentive condition, participants were given verbal encouragement from the experimenter, both at outset and during the trials, to try and perform the task optimally. In the financial incentive condition, participants were informed that they could win 1p or 10p per correct trial. We expected antisaccade performance to be better in incentive conditions. In line with previous research (e.g. Duka & Lupp 1997; Hardin et al., 2007; Jazbec et al., 2006), we predicted that participants would make fewer antisaccade errors when receiving financial incentive. Furthermore, we expected to see a difference in antisaccade errors between the no incentive and the verbal incentive group, as encouragement has the potential, as a secondary re-enforcer, to improve performance. Based on the findings of Jazbec et al. (2006) and Hardin et al. (2007) a further prediction was that correct antisaccade latencies would be reduced under incentive conditions.

Experiment 1a

Method

Participants

Participants comprised 58 undergraduate students from the University of Sussex with normal or corrected to normal vision, of which 46 were female and 12 male. Ages ranged from 18 – 31 years (M = 24.6, SD = 3.87). Participants took part as part of their course requirements. All participants received course credit, with some also obtaining monetary reward, depending on which condition they were allocated to. Participants were naïve to the purposes of this study. All participants provided written consent and the study was approved by the relevant ethics committee.

Apparatus

Eye movements were recorded using an Eyelink II eye tracker (SR-Research Ltd., Ontario) with a temporal resolution of 2 ms and a spatial resolution of around 0.25 degs. The stimuli were displayed on a 21inch CRT monitor with a screen resolution of

1280 x1024 pixels and a refresh rate of 100Hz. Actual screen dimensions were 40cm horizontal and 30cm vertical. Participants were seated approximately 70cm from the screen in an adjustable chair that had been modified to prevent any rotational movement. Each set of trials was preceded by a calibration procedure, during which participants focused their eye gaze on 9 separate targets in a 3 x 3 grid. Only right eye movements were recorded.

Stimuli

For each trial the display comprised a white background containing a red circle (0.25 degrees in diameter) as the central fixation point. After a brief interval (800-1200) the fixation circle disappeared from the screen and following a 200msec gap, was replaced by the target stimulus (red circle, also 0.25 degrees in diameter), either +/- 4 or 8 degrees from the centre point. The target stimulus was displayed for 2000msec, which was enough time for a participant to initiate a saccade towards or away from it. However if a saccade was made that reached the goal location, the target would remain on screen for a further 300msec.

Design

In a between-participants design, participants were randomly allocated to one of three different incentive conditions, (no incentive, verbal incentive, financial incentive). The Saliency of trial value was explored by comparing antisaccade performance on 1p and 10p trials. Each participant completed 64 trials and no practice trials were given.

Procedures

Standard antisaccades (no incentive)

In the no-incentive condition, participants fixated on a central stimulus (a small red circle) that was replaced by a target at +/- 4 or 8 degrees from the central point. Participants were instructed to look to the mirror image location of the sudden onset target as quickly and as accurately as possible. A total of 64 trials divided into 4 blocks of 16 were completed with no practice trials included.

Antisaccades with verbal incentive

The stimulus properties were identical to the block of standard antisaccades described above. However, the experimenter provided verbal encouragement to participants, to perform well at the outset of the experiment, and at regular occasions during performance. Examples of the verbal encouragement provided during the trials included phrases such as “well done”, or “keep trying to make sure you don’t look at the target.”

Antisaccades with financial incentive

The procedure of this condition was identical to the previous two conditions except participants were informed that they would be given either 1p or 10p per correct trial. The potential “prize” was indicated on the computer screen at the start of each trial and at the end of each trial participants were informed as to whether they had made a correct antisaccade or an antisaccade error. After a correct trial, a positive message was shown on screen in green font (“well done keep it going”) and following an error trial a negative message was shown in red font (“bad luck keep trying”). The subtotal “earnings” were updated at 4 intervals (after every 16 trials). An equal number of trials could earn the participant either 10p or 1p, resulting in a total possible earnings of £3.52.

Measures

Measures included the number of antisaccade errors (saccades made after target onset towards the sudden-onset target) and these were counted and expressed as a percentage of the total number of valid trials performed. Correct latency (difference in milliseconds between the target onset and the onset of a saccade made to the opposite hemifield (without any intervening erroneous saccade) was also measured. Additional measures were recorded, including correct amplitude, latency to correct an antisaccade and error latency, but in an exploratory analysis, incentive did not affect these variables, therefore for simplicity they are not reported here. Trials were excluded from analysis if a) if the eye was not within 40 pixels of the central fixation point at the time of target appearance; b) the primary saccade was made less than 0msec after target onset or more than 600ms after target appearance (delayed); c) no saccade was made within the trial duration, or the primary saccade was obscured by blinks. As a result of this criteria, 7% of data was excluded.

Data analysis

Eye movement recordings were visualized and quantified off-line using Data Viewer software (SR-Research, Ontario). Saccade onset time was defined as the first of a series of three data points for which the instantaneous velocity exceeded 30◦/s. Similarly the end point of the movement was defined as the first of three data points following saccade onset for which the instantaneous velocity fell below 30◦/s. Bonferonni corrections applied to all contrasts.

Results

Percentage of antisaccade errors

Percentage of antisaccade errors, across incentive condition is plotted in figure 2.1. A one way between-participants ANOVA was conducted, looking at the effects of incentive group on antisaccade errors.

There was no significant main effect of incentive on antisaccade percentage of errors, ($F(2, 55) = 1.49$, $p = .23$, $r = .16$).

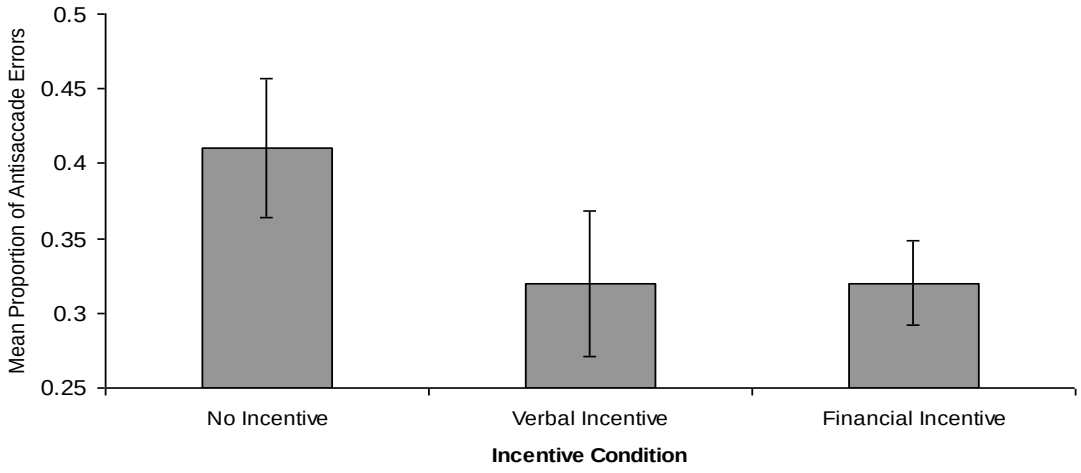

Figure 2.1. Antisaccade error rate across incentive groups

Correct antisaccade latencies

Correct antisaccade latencies across incentive conditions are plotted in figure 2.2. A one way between-participants analysis of variance was conducted, looking at the effects of incentive group on correct antisaccade latencies.

There was a significant main effect of incentive group on correct antisaccade latencies ($F(2, 55) = 3.73$, $p = .03$, $r = .25$). Participants were significantly faster to make correct antisaccades in the financial incentive condition than in the no incentive condition ($t(55) = 2.66$, $p = .03$, $r = .34$). There was no difference in correct latencies between the no incentive condition and the verbal incentive condition ($t(55) = .75$, $p = 1.0$, $r = 0$). Finally, there was no difference between the verbal incentive condition and the financial incentive condition ($t(55) = 1.93$, $p = .17$, $r = .25$). Taken together, the results imply that correct antisaccade latencies were more sensitive to incentive than antisaccade errors.

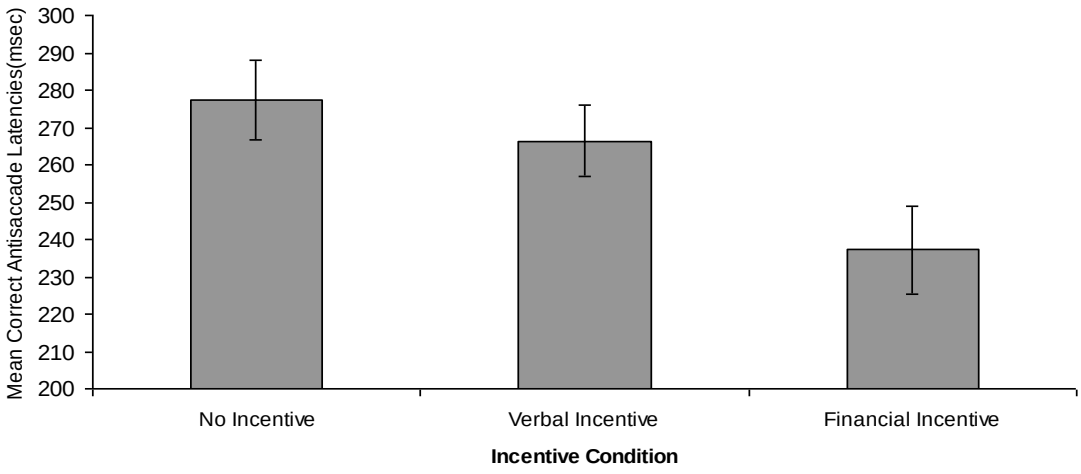

Figure 2.2. Correct antisaccade latencies across incentive groups

Saliency

There was no effect of saliency on either errors or correct latencies.

Discussion

In this first study, we explored the effects of incentive on antisaccade performance, by giving participants no incentive, a verbal incentive, or a financial incentive. Neither incentive had a significant effect on antisaccade errors. However, participants were faster to make a correct antisaccade in the financial incentive group compared to those in the no incentive group.

The finding that correct antisaccade latencies were significantly reduced when participants were given a financial incentive is in line with Hardin et al. (2007), but not with Blaukopf & Di Girolamo (2005, 2006); Duka & Lupp (1997); Jazbec et al. (2005, 2006). In order to maximize the valence of the financial incentive, participants also received trial by trial feedback on their performance. In other words our financial incentive condition confounds incentive with feedback, as participants knew after each trial whether they had made a correct or incorrect response, on the basis of the reward they did or did not receive. Previous research has shown that participants can be unaware of as many as 50 ± 25% of their antisaccade errors (Mokler & Fischer, 1999). Consequently, receiving feedback regarding task performance could be beneficial. Models of cognitive control (Miller & Cohen, 2001) argue that on a given task, participants will monitor their performance and adjust accordingly, for example increasing control to ensure accuracy. Therefore, a manipulation that increases participants' awareness of their antisaccade performance could improve antisaccade performance independently of any effect of incentive.

In terms of antisaccade errors, we failed to replicate the findings of Duka & Lupp (1997), Jazbec et al. (2006) and Hardin et al. (2007), as incentive did not reduce antisaccade errors. However our error rate results support Blaukopf & Di Girolamo (2005, 2006) who also failed to observe an effect of incentive on antisaccade errors. One explanation for the lack of effect was that the amount of monetary reward given was simply not large enough to modulate performance to the extent that error rates were altered. This was supported by the fact there was no saliency effect (no difference between 1p vs. 10p trials).

It is interesting to note that according to parallel programming models of antisaccade performance (Massen, 2004; Nieuwenhuis et al., 2004; Reuter & Kathmann, 2004) a reduction in correct antisaccade latency should result in a reduction in antisaccade errors. However, a number of researchers have argued that in some circumstances correct antisaccade latency may be a more sensitive index than antisaccade error rate (e.g. Hutton & Ettinger, 2006; Rycroft et al., 2007). We observed a reduction (from 41% to 32%) but this did not reach significance.

In the following experiment, we aim to determine whether feedback alone or financial incentive alone are capable of reducing correct antisaccade latencies.

Experiment 1b

Two further conditions were created. One condition simply provided participants with trial by trial feedback (audio + visual) as to whether they had made a correct antisaccade or an error, and the second provided financial incentive, without any trial by trial feedback as participants were only told how much they had earned at the end of the experiment. In the feedback alone condition, we increased the amount of feedback given, as an audio signal was sounded in response to the outcome of each trial. In order to establish whether a greater financial incentive might also result in a significant reduction in antisaccade error rate as well as correct antisaccade latency, we increased the amount of monetary reward that could be won. Participants could now win 10p for each trial, which was an improvement from the first study and equivalent to the amount used in previous research (Blaukopf & Di Girolamo, 2005). A further justification for using a flat rate of 10p per trial was that there was no saliency effect (1p vs. 10p) found in our first study.

In order to maximize the possibility of observing an effect on error rates, we created a condition which maximized trial by trial feedback by giving both an audio and visual signal. The audio signal was a positive game show sound if correct, and a negative game show sound if incorrect. The visual signal was “well done keep it going” for a correct response and “bad luck keep trying” for an incorrect response. Performance in these two conditions were compared to the data from experiment 1a in order to determine whether financial incentive alone was sufficient to reduce correct antisaccade latencies, or whether trial by trial feedback on its own was also sufficient. We predicted that groups who received an incentive would make significantly less antisaccade errors than the no incentive group. Based on the first study, we expected the financial incentive group to have the fastest correct antisaccade latencies.

Method

Participants

A further 30 people were recruited for the study, making the combined sample (N = 88). The new participants were 8 male and 22 female students from the University of Sussex aged between 19 and 40 (M = 26.20, SD = 4.36). All participants had normal

to corrected normal vision and were tested individually. Three participants took part as part of their course requirements and the remaining 27 were post graduate volunteers. 15 of the students were given a monetary reward, depending on their group and performance. Testing lasted about 15 minutes.

Apparatus and stimuli

The apparatus and stimuli used were identical to the first experiment.

Design

A between-participants design was deployed as participants were divided into two groups, feedback alone, or financial incentive alone. Participants completed one block of 64 antisaccades and no practice trials were given.

Procedures

Standard antisaccades (no incentive)

This condition was identical to the no incentive condition in the previous study.

Antisaccades with feedback (alone)

Participants performed 64 standard antisaccade trials, however after each trial, feedback on their accuracy was given, both visually and in an auditory fashion. The visual feedback consisted of a message of "bad luck keep trying", or "well done keep it going", depending on if they had made an error or not. The auditory feedback comprised of a negative or positive game show sound (60db), again depending on their accuracy.

Antisaccades with financial incentive (alone)

Participants performed 64 standard antisaccade trials (with no feedback on performance). Before starting the task, participants were informed that they would be rewarded with 10p for each trial they performed correctly, and thus could earn a maximum of £6.40. Participants received no feedback as to their performance after each trial.

Measures and data analysis

Measures and data analysis were identical to those in the first study and resulted in the exclusion of 6% of data.

Results

The two conditions from this experiment were combined with the three conditions from the first experiment and analyzed together so as to compare the current incentive conditions to the no incentive condition from the first experiment.

Percentage of antisaccade errors

Percentage of antisaccade errors as a function of incentive group is illustrated in figure 2.3. A one way between-participants ANOVA was conducted, looking at the effects of incentive group on antisaccade errors.

Despite the between condition differences in error rate apparent in Figure 2.3, the ANOVA revealed no main effect of incentive on antisaccade errors ($F(4, 83) = .86$, $p = .49$, $r = .10$). The size of the error bars suggests this lack of a significant main effect is due primarily to the large variances in antisaccade error rate within each group.

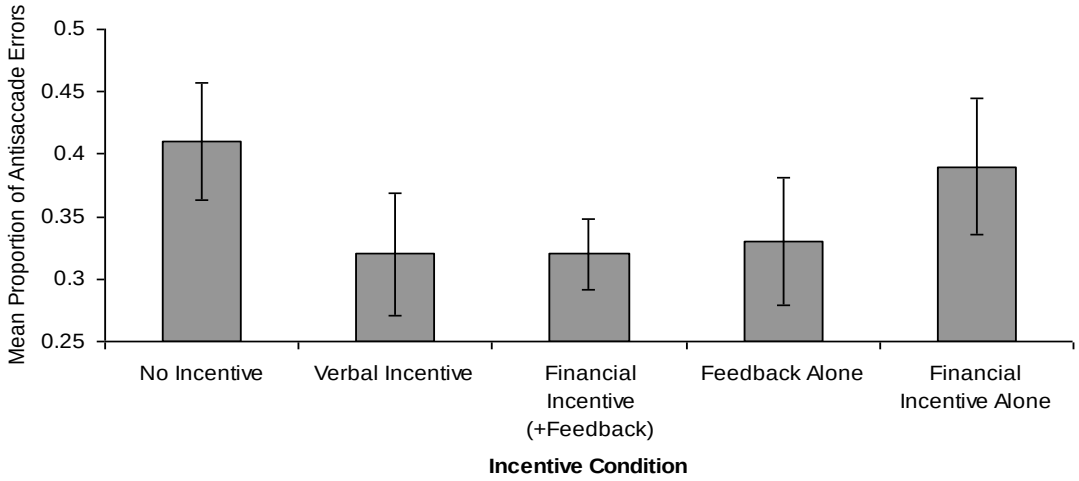

Figure 2.3. Antisaccade error rate across incentive groups

Correct antisaccade latencies

Correct antisaccade latencies across incentive conditions are plotted in figure 2.4. A one way between-participants analysis of variance was conducted, looking at the effects of incentive group on correct antisaccade latencies.

The one way analysis of variance revealed a significant main effect of group on correct antisaccade latencies $F(4, 83) = 4.30$, $p < .01$, $r = .22$. Paired t-tests showed that participants were significantly faster in the feedback alone condition compared to the no incentive condition ($t(33) = 2.62$, $p = .01$, $r = .41$). Participants were also significantly faster in the financial incentive alone condition, compared to the no incentive condition ($t(33) = 3.44$, $p = .01$, $r = .51$). There was no difference in correct antisaccade latencies between the financial incentive (+feedback) condition and the feedback alone condition ($t(31) = .28$, $p = .79$, $r = .05$). Similarly, there was no difference in correct latencies between the financial incentive (+feedback) condition and the financial incentive alone condition ($t(31) = .80$, $p = .43$, $r = .14$). An additional comparison revealed that participants were significantly faster in the feedback alone condition compared to the verbal incentive condition ($t(33) = 2.10$, $p = .04$, $r = .34$), suggesting that increased amounts of feedback had a positive effect on correct latencies. In sum, giving participants feedback alone and financial incentive alone have reduced correct antisaccade latencies compared to no incentive.

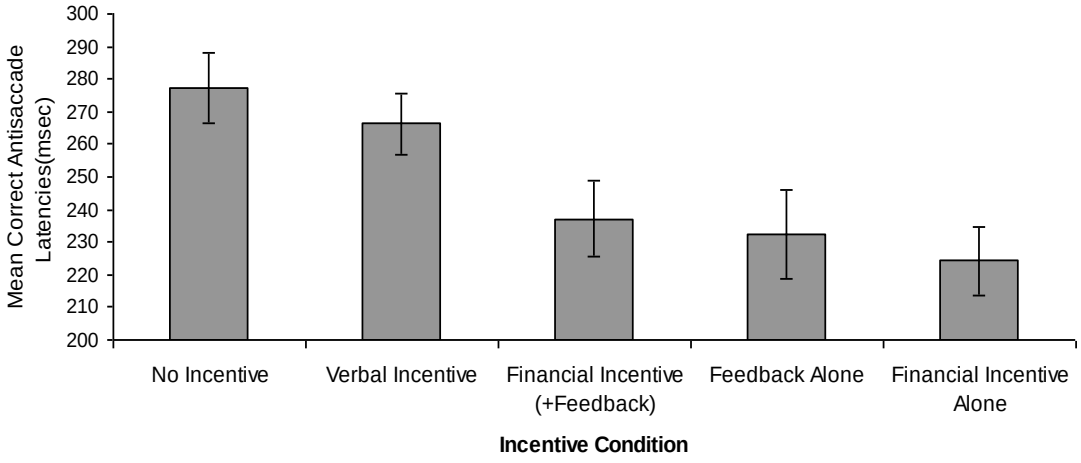

Figure 2.4. Correct antisaccade latencies across incentive groups

Discussion

The current experiment set out to tease apart the potential confound of the financial incentive condition in experiment 1a, in which financial incentive was accompanied by trial by trial feedback on performance. We found that, compared to the no incentive condition in experiment 1a, both feedback alone in the absence of a financial incentive, and financial incentive alone in the absence of trial by trial feedback were capable of reducing correct antisaccade latencies. These results support those of Hardin et al. (2007), who also found that financial incentives can lead to faster correct antisaccades and clarify that this effect can occur independently of any effect on performance of trial by trial feedback.

The finding that feedback alone can improve correct antisaccade latencies is novel and supports recent models of cognitive control (Miller & Cohen, 2001). Such cognitive control accounts argue that participants monitor their ongoing behaviour during task performance and make behavioural adjustments accordingly. In the present study, it was likely that participants were using trial by trial feedback to monitor their ongoing performance on the task. By being made aware of when errors occur, participants will increase levels of cognitive control (activity in neural pathways supporting correct task performance), resulting in a reduction in correct latencies. Tatler & Hutton (2007) found evidence that participants use information about previous antisaccade trial outcome (i.e. error or correct saccade), for use on current antisaccade trial performance. The authors found that there was an increased probability of making an error (erroneous prosaccade) on the current trial if the previous trial was also an error. In addition, after a slowly corrected error (> 150msec) correct antisaccade latencies were longer than after correct antisaccades. This supports the 'post error slowing' strategy proposed by Botvinick et al. (2001) who suggested that participants slow their performance on the current trial, if the previous trial was an error. However, Tatler & Hutton (2007) only found 'post error slowing' when the previous trial error was corrected slowly and found 'post error quickening' when the previous trial error was corrected quickly (< 80msec). The authors suggested that 'post error slowing' is more likely to occur on correct antisaccades that followed slowly corrected errors because participants are more aware of these errors. Their results suggest participants use information on previous trials to monitor ongoing behaviour on current trials.

As in experiment 1a, despite differences in error rate of between 5 and 9% between conditions, there was no significant main effect of incentive. Error rates were unusually high across experiments 1a and 1b. Large scale studies of healthy participants typically observe antisaccade error rates ~ 20% (e.g. Ettinger et al., 2003, 2005; Tatler & Hutton, 2007). In contrast, our standard no incentive condition from experiment 1a showed 41% error rate. The variance in error rates was also notably high within all conditions in both experiment 1a and 1b. Although it is not uncommon to find large variability in antisaccade performance (e.g. 0 -100% error rates Smyrnis et al., 2002), it is not clear why our average error rates were so high. One possible explanation was that participants were not abundantly clear on what exactly constituted an antisaccade error. Although instructions were explicitly stated, it is difficult to know if this sample was unaware that a glance to the target stimulus resulted in an error. In addition, participants quite simply may have been unmotivated, consequently affecting performance. This may have occurred because some of the participants were simply concerned with fulfilling their requirement to collect course credits by participating in experiments.

The competitive race account outlined in the introduction predicts that any manipulation which results in faster correct antisaccade latencies ought also to result in a reduction in error rates. In the next experiment we combined incentive with trial by trial feedback and used a within-participants design in order to replicate our previous findings on antisaccade latency, and establish whether a significant reduction in error rate might also be observed using an experimental design in which large variances in error rate within groups are not an issue.

Experiment 2

In the final study, we again explored the effects of incentive on antisaccade performance. The previous studies found that financial incentive and trial by trial feedback (both alone or combined) led to reductions in correct antisaccade latencies. Verbal encouragement, financial incentive with feedback, and feedback alone also led to small reductions in antisaccade error rate, but these reductions were not significant. The level of reductions (5-9%) were however, of equivalent magnitude or even greater than those observed in other studies. For instance, a smaller difference in errors of 3% between antisaccades with no incentive and antisaccades with incentive was statistically

significant in a within-participants design used by Duka & Lupp (1997). Similarly, Jazbec et al. (2005) observed a 3% significant difference in antisaccade errors between neutral trials and reward trials in healthy participants using a within-participants design. To find an effect of incentive on antisaccade errors, and to remove the influence of large variations in antisaccade error rate within groups, we incorporated a within-participants design.

In experiment 1b, both feedback alone and financial incentive alone reduced correct latencies, but only feedback alone reduced antisaccade errors (all be it not significantly). This would suggest that error rate is more sensitive to feedback, but there is a possibility that providing participants with both feedback and financial incentive will alter the number of antisaccade errors made. Therefore, combining both types of incentive (feedback and financial) means that participants will receive the maximum incentive and error rates could be reduced as a result of this additive effect.

We compared antisaccade performance in three different conditions, no incentive, financial incentive + audio/visual feedback and financial incentive alone. We predicted that antisaccade errors would be lowest in the financial incentive + audio/visual feedback condition.

Method

Participants

Participants comprised 30 students from the University of Sussex, of whom 18 were female and 12 male. Ages ranged from 20 – 44 years (M = 27.4, SD = 5.22) and all participants had normal to corrected normal vision. Data from one participant was not included in the analysis as they made an unusually large number of anticipatory eye movements in the reward + feedback condition, making the sample 29. In the advertisement, participants were informed that they would receive £4 for taking part in the study, but were told at the testing session that they could keep the amount of money they won. If they won less than £4, they would still get £4. Participants were naive to the purpose of the study. All participants provided written consent and testing lasted approximately 25 minutes.

Apparatus and stimuli

The apparatus and stimuli used were identical to the previous experiments.

Design

A within-participants design was used, with three separate conditions: no incentive, financial incentive + audio/visual feedback, financial incentive alone. Stimulus parameters used for the financial incentive + audio visual feedback condition and for the financial incentive alone condition were the same as previous conditions from experiment 1b. The financial incentive alone condition was identical to the financial incentive condition from experiment 1b. Each participant completed a total of 192 trials divided into 3 blocks of 64 and the order in which the three conditions were performed was fully counterbalanced.

Procedures

Standard antisaccades (no incentive)

The procedure of this condition was identical to the no incentive conditions from the previous two experiments.

Antisaccades with financial incentive + audio/visual feedback

Participants had to complete a block of antisaccades where they were rewarded with 10p if they made a correct antisaccade. They were given visual feedback after every trial as to whether they had made a correct antisaccade ("well done keep it going") or an antisaccade error ("bad luck keep trying") and their subtotal winnings. In addition, feedback for a correct trial was accompanied with a positive "game show" sound (60db) and an error trial was accompanied with a negative "game show" sound. No practice trials were given and the total possible jackpot was £6.40.

Antisaccades with financial incentive alone

This condition was identical to the financial incentive alone condition from the previous experiment.

Measures and data analysis

These were identical to the previous experiments and resulted in the exclusion of 5% of data.

Results

Percentage of antisaccade errors

A one way within-participants ANOVA was conducted on the percentage of antisaccade errors made. The ANOVA revealed a main effect of incentive condition on antisaccade error rate ($F(1.72, 48.06) = 3.29$, $p = .05$, $r = .25$, Huynh-Feldt correction, $e = .86$, see figure 2.5). Paired t-test comparisons showed that participants made significantly fewer antisaccade errors in the financial incentive (+audio/visual feedback) condition compared to the no incentive condition ($t(28) = 2.02$, $p = .05$, $r = .36$). However, there was no difference in antisaccade errors when participants were given financial incentive alone compared to no incentive ($t(28) = .42$, $p = .68$, $r = .08$). Finally, participants made significantly fewer antisaccade errors in the financial incentive +audio/visual feedback condition compared to the financial incentive alone condition ($t(28) = 2.59$, $p = .05$, $r = .44$).

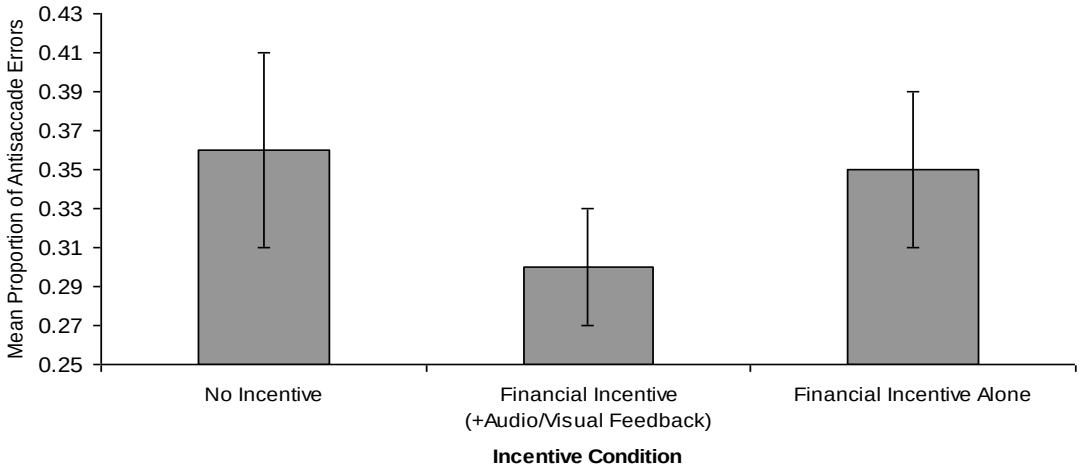

Figure 2.5. Antisaccade error rate across incentive conditions

Correct antisaccade latencies

A one way within-participants ANOVA was conducted on correct antisaccade latencies. The ANOVA found a main effect of incentive on correct latencies ($F(2, 56) = 3.85$, $p = .02$, $r = .25$). Paired t-tests revealed that participants were not significantly faster in the financial incentive +audio/visual feedback condition compared to the no incentive condition ($t(28) = 1.12$, $p = .27$, $r = .21$). However, participants were significantly faster to make a correct antisaccade when given financial incentive alone compared to no incentive ($t(28) = 2.86$, $p < .01$, $r = .48$). Finally, there was no difference in correct antisaccade latencies between the two incentive conditions ($t(28) = 1.65$, $p = .11$, $r = .30$). The data were reanalyzed, removing one participant with outlying correct latency (more than two standard deviations above the group mean) and two other participants who made >75% errors across all three conditions (resulting in comparatively few correct trials over which to average the latencies). With these participants removed, the difference between the no incentive condition and the combined financial incentive + feedback condition approached significance ($t(25) = 1.7$, $p = .10$, $r = .32$). The difference between the no incentive and financial incentive alone condition remained highly significant ($t(25) = 3.4$ $p < .01$, $r = .56$).

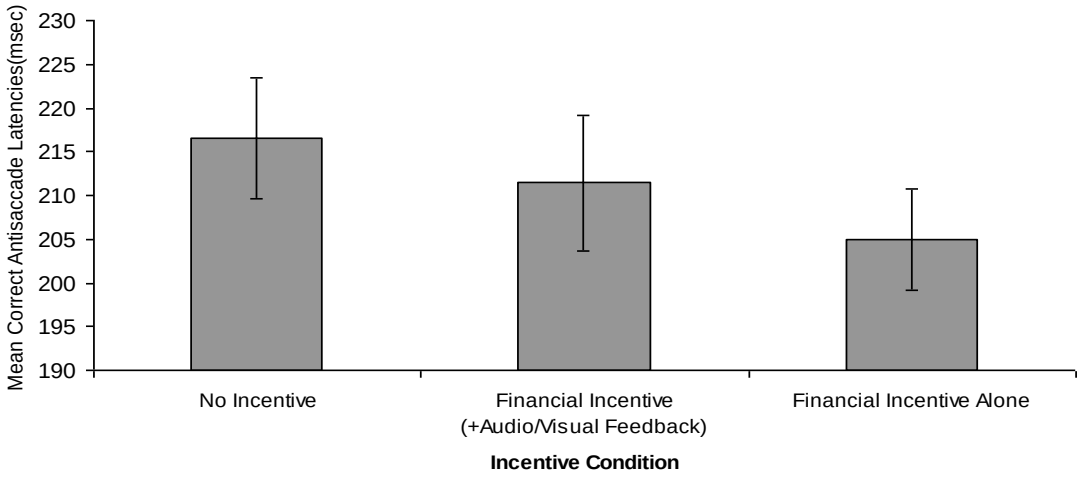

Figure 2.6. Correct antisaccade latencies across incentive conditions

Discussion

In the final experiment we explored the effects of financial reward (both alone and combined with trial by trial feedback) on antisaccade performance. Receiving a financial reward alone did not reduce errors compared to receiving no incentive. However, the combination of financial reward and trial by trial audio/visual feedback did result in a significant decrease in antisaccade error rate (the lowest error rate found across the three experiments). Surprisingly, the latency data showed a different pattern, as financial incentive alone reduced correct antisaccade latency, but financial incentive combined with trial by trial audio/visual feedback did not.

The results of experiment 2 confirm the general findings of the previous two experiments that both financial incentive and trial by trial feedback can impact on antisaccade performance, but raises important questions concerning the relationship between antisaccade error rate and correct antisaccade latency. In the general discussion that follows, the present results are considered in light of the findings of the previous experiments, and their implications for models of antisaccade performance are discussed.

General Discussion

We explored the impact of top-down factors on antisaccade performance across a series of experiments by manipulating the incentive to make correct responses. In experiments 1a and 1b , using a between-participants design, we found that providing participants with verbal encouragement, financial incentive combined with trial by trial feedback, or trial by trial feedback alone all reduced antisaccade error rate between 5 and 9%. However, these reductions were not significant due to large variances in antisaccade error rate within groups. The effects of incentive on correct antisaccade latencies were more robust, and financial incentive combined with trial by trial feedback, and both trial by trial feedback alone and financial incentive alone all resulted in significant reductions in correct antisaccade latencies. In sum, giving participants feedback alone and financial incentive alone (exp. 1b) improved correct antisaccade latencies to the same magnitude as when they were combined (exp. 1a). Despite the fact that the reductions in error rate were not statistically significant, it is of interest to note

that contrary to parallel programming accounts, there was no obvious relationship between the effects of the different incentives on correct latency and antisaccade error rates. For example, verbal encouragement, which appeared to lower error rates had no effect on correct latencies, and financial incentive alone (which had only a moderate impact on error rate) resulted in the greatest reduction in correct antisaccade latency. The implications of these findings are considered in the following discussion.

In experiment 2, a within-participants design was used to determine whether incentives might result in a statistically significant reduction in error rate under conditions in which large variances in error rate within groups were not a factor. Antisaccade errors were only significantly reduced when financial incentive was combined with trial by trial feedback, whereas correct antisaccade latencies were only reduced in the financial incentive alone condition. Again, these findings are problematic for parallel programming accounts of antisaccade performance, which clearly predict that a manipulation that results in a reduction in correct antisaccade latency should also reduce antisaccade error rate.

Given the lack of consistency in terms of the effect of incentive on antisaccade performance in previous studies, it is disappointing, but perhaps unsurprising, that a similarly confused picture has emerged from these studies. In all three experiments, we found an effect of incentive on correct latencies. The only other study that found an effect of incentive on antisaccade latencies was Hardin et al. (2007). Interestingly, like the present studies, Hardin et al also compared the effects of incentives within blocks of antisaccade trials. In all the other studies, mixed blocks were employed, in which antisaccades were interspersed with prosaccades, and a central cue indicated which type of saccade was to be performed at the beginning of each trial. One possible explanation of these findings is that within a block of antisaccade trials, the effect of incentive is more powerful, because participants are better able to maintain and focus their concentration on performing an antisaccade as quickly and as accurately as possible.

The effect of incentive on antisaccade error rate in past research is inconsistent. Reductions in errors have been found in some studies (Duka & Lupp, 1997; Jazbec et al., 2005, 2006; Hardin et al., 2007) but not others (Blaukopf & Di Girolamo 2005, 2006). We observed no reduction in error rate in experiments 1a and 1b, and a significant reduction when financial incentive was combined with trial by trial feedback in a within-participants design in experiment 2. As argued in previous discussions, in experiments 1a and 1b, error rate was unusually high, and variance within the groups

was also very high, both of these factors reduced the chances of any observed reductions being statistically significant. Previous research using within-participants designs, found small but significant differences in average error rates between incentive and non-incentive conditions (Duka & Lupp, 1997; Hardin et al., 2007; Jazbec et al., 2005, 2006). It is possible that using a between-participants design prevented us from finding an effect of incentive on antisaccade errors in experiments 1a and 1b because of within group variance. In experiment 2 we changed the design to within-participants to alleviate the effects of within-group variance from our previous experiments and this resulted in reduced errors in the financial incentive with audio/visual feedback condition.

One somewhat unexpected feature of the present results is the relative lack of consistency between the effects of any given condition on error rate and correct antisaccade latency. In experiments 1a and 1b, verbal encouragement resulted in (non significantly) reduced error rate, but no change in correct antisaccade latency, whereas financial incentive alone significantly reduced correct antisaccade latency, but had no impact on error rate. Similarly, in experiment 2, financial incentive alone reduced correct antisaccade latency, but had no impact on error rate. These findings are not easy to accommodate within current parallel programming models of antisaccade performance (Massen, 2004; Reuter & Kathmann, 2004).

Parallel programming accounts of antisaccade performance argue that target onset leads to increased activity in separate prosaccade and antisaccade pathways that "compete", with the winner reaching the threshold for triggering a saccade first. Massen (2004) argued that a manipulation capable of significantly reducing correct antisaccade latencies (without reducing correct prosaccade latencies) should significantly reduce antisaccade errors, as antisaccade activity is augmented, increasing the likelihood of activity in this pathway reaching the threshold for saccade triggering before activity in the erroneous prosaccade pathway. In the present study we did not measure prosaccade performance, thus it is difficult to say if our results can be accommodated by parallel programming models of antisaccade performance. We found incentive reduced correct antisaccade latencies and Watanabe et al. (2003) found incentive reduced correct prosaccade latencies in non-human primates. Hence it is possible that both prosaccade and antisaccade latencies could be influenced by incentive to the same degree, which according to Massen (2004), would not produce a change in antisaccade errors. Jazbec et al. (2006) measured the effects of incentive on both prosaccades and antisaccades in

their study. However, their results also do not support parallel programming accounts. They found that for healthy adults, antisaccade errors were reduced by incentive even though correct prosaccade and correct antisaccade latencies were both reduced (all be it not significantly) by 3msec. This contradicts parallel programming accounts which suggest that prosaccade and antisaccade latencies must be differentially affected for a reduction in antisaccade errors to occur. If a manipulation increases activity in the antisaccade pathway only, then this should increase the likelihood of a correct antisaccade reaching the threshold for triggering a saccade before an erroneous prosaccade. Future work will benefit from investigating the impact of incentive on prosaccade performance to test specific predictions from parallel programming accounts.

An important finding of these experiments was that providing participants with non-financial incentives (e.g. trial by trial feedback) can impact on antisaccade performance. Receiving feedback on performance was a contributing factor in several of our conditions. Providing trial by trial feedback alone (experiment 1b) regarding the outcome of the trial, was sufficiently motivating to also improve correct response latencies. In experiment 1b, audio/visual feedback reduced antisaccade errors more than financial incentive and in the final experiment, the condition containing feedback also reduced errors more than the financial incentive alone condition. Participants were also updated on their subtotal after each trial, which may have been a contributing factor. This suggests that participants were using feedback to monitor and adjust performance.

Models of cognitive control (Botvinick et al., 2001; Dehaene et al., 2003; Miller & Cohen, 2001) suggest that in tasks concerned with cognitive control, participants use a conflict monitoring procedure. Monitoring of ongoing performance becomes crucial when a prepotent response has to be over-ridden (e.g. avoid making prosaccade to target). Evidence from a range of sources suggests that such trial by trial modulation of performance takes place (Tatler & Hutton, 2007). Cognitive control models argue that our response to the outcome of the previous trial may impact on current trial performance. These models suggest that a ‘post error slowing’ strategy is adopted, whereby the participant increases control to ensure accuracy, by slowing their response on a trial that followed an error trial. Giving feedback after each trial could enhance performance, as participants will know if they were performing correctly then decide if they need to increase cognitive control on the upcoming trial. Feedback may be important to the antisaccade task because it improves awareness of errors and

participants can use this awareness to improve their antisaccade performance, especially as a large proportion of errors are often unidentified (Mokler & Fischer, 1999).

There are several ways in which the experiments reported here might have been improved. Firstly, if prosaccade measures are incorporated, then we will know if specific predictions outlined by parallel programming accounts will be met (i.e. will incentive differentially impact on prosaccade and antisaccade latencies?). It is possible that the amount of potential earnings per correct trial was not sufficient to genuinely motivate participants. Although we increased the potential earnings from 1p or 10p per correct trial in the first experiment to 10p per correct trial in the second experiment, our study paid out less compared to previous work (Blaukopf & Di Girolamo, 2006; Hardin et al., 2007 Jazbec et al., 2005, 2006). Increasing the potential earnings per trial may impact on antisaccade performance.

In conclusion, we have shown that financial and non-financial incentives can have modest effects on antisaccade performance. However, in line with past research, the results of the present study were inconsistent. We have however shown that non-financial incentives can impact on correct antisaccade latencies in healthy participants. The fact that providing feedback can improve antisaccade performance is important, as it suggests that participants are using the feedback as a cognitive control mechanism to monitor and adjust their antisaccade performance. Our results suggest that incentives can influence the programming of volitional eye movements, particularly in relation to the programming of the response time of a correct antisaccade. Further research is required to clear up the inconsistencies of the effects of incentives on antisaccade performance. Neuroimaging studies would be wise to adopt antisaccade paradigms to determine the top-down mechanisms responsible for incentive related modulation of saccadic eye movements.

Article 3 – The Role of Different Task Instructions on Pro and Antisaccade Performance

A revised and abbreviated version of this paper has been published as: Taylor, A. J. G., & Hutton, S. B. (2009). The effects of task instructions on pro and antisaccade performance. *Experimental Brain Research, 195,* 5-14.

Abstract

In the antisaccade task participants are required to overcome the strong tendency to saccade towards a sudden onset target, and instead make a saccade to the mirror image location. The task thus provides a powerful tool with which to study the cognitive processes underlying goal directed behaviour, and has become a widely used index of "disinhibition" in a range of clinical populations. Across three experiments we explored the role of top-down strategic influences on antisaccade performance by varying the instructions that participants received. In the first two experiments, instructions to delay making an antisaccade resulted in a significant increase in correct antisaccade latencies and a reduction in erroneous prosaccades towards the target. Instructions to make antisaccades as quickly as possible resulted in faster correct antisaccade latencies whereas instructions to be as spatially accurate as possible increased correct antisaccade latencies. Neither of these manipulations resulted in a significant change in antisaccade error rate. In the second experiment, instructions to delay making a prosaccade towards the target resulted in increased correct latencies on a prosaccade task. Instructions to make a prosaccade as quickly as possible and as accurately as possible did not affect correct prosaccade latencies. In a third experiment, participants were faster under delayed prosaccade instructions than delayed antisaccade instructions and made fewer direction errors in delayed pro and antisaccade tasks than in a standard antisaccade task. The implications of these results for current models of antisaccade performance, and the interpretation of antisaccade deficits in clinical populations are discussed.

Introduction

The antisaccade task requires participants to refrain from looking at a sudden onset target and direct their gaze instead to the exact opposite location. Antisaccade error rate (the percentage of erroneous prosaccades made towards the target) is often used as a measure of "disinhibition" in clinical settings, and a large number of studies have described increased errors in various psychiatric and neurological populations (see Hutton & Ettinger, 2006; Hutton, 2008 for reviews). In contrast to the extensive body of research describing impaired antisaccade performance in patient populations, there has been comparatively little research into the cognitive processes that underlie successful antisaccade performance in healthy participants. Average error rates in healthy participants are typically around 20%, but this figure can vary considerably across studies and also across individuals (e.g. Evdokimidis et al., 2002, average error rate = 23%, range 0-100%, Mokler & Fischer, 1999, average error rate = 19%, range 0-60%, Tatler & Hutton, 2007, average error rate = 24.1, *SD* = 8.2). It is only recently that researchers have begun to address the possible sources for this variability (e.g. Taylor & Hutton, 2007). In this paper we investigate the extent to which antisaccade performance can be modified by varying the nature of the instructions healthy participants receive, and whether such effects are predicted by current theoretical models of the task.

Whilst saccades are often thought of as relatively automatic and stimulus driven events, there is a large body of research demonstrating that fundamental properties of saccades such as their latency and spatial accuracy can be influenced by a range of "cognitive" factors (see Hutton, 2008 for a review). In an interesting study, Machado & Rafal (2000) investigated the extent to which participant's "readiness" to make saccades influenced saccade latencies. They found that participants made faster prosaccades towards peripheral targets in blocks where the target appeared in 80% of trials compared to blocks in which it appeared in 20% of trials. In addition, the gap effect was reduced in the high frequency (80%) target blocks. Similar effects were observed when participants made endogenously driven saccades on the basis of a tone signal. The authors argued that participants respond strategically to the manipulation of saccade frequency, and in situations in which saccades are required frequently they inhibit fixation neurons in the Superior Colliculus (SC) thus disinhibiting collicular movement cells and decreasing prosaccade latencies. Similarly, in situations in which saccades are

required on the minority of trials, activity in fixation neurons remains strong, thus inhibiting activity in the movement cells and thereby increasing the average latency of prosaccades. These findings were recently replicated, and extended to the antisaccade task (Van Koningsbruggen & Rafal, 2008).

Trottier & Pratt (2005) demonstrated that prosaccade latencies can be reduced markedly simply by requiring participants to determine whether the central pixel of the peripheral target was displaced or not. This effect of task instructions has recently been replicated (Guyader, Malsert, & Marendaz, 2008). In this experiment participants were faster to make saccades towards targets under "identify" instructions when they had to determine whether the target was a 6 or a 9 compared to "glance" instructions when they simply had to look towards a target as quickly as possible. Importantly, this effect did not to occur when participants were instructed to make antisaccades. The authors argued that the effect does not occur in the antisaccade task as the cognitive processes required to make the antisaccade compete with higher level cognitive processes required to perform the identification task.

Mosimann, Felblinger, Colloby, & Müri (2004) also provide evidence that prosaccade metrics can be readily influenced by strategic factors. They required participants to make saccades under various task instructions. When participants were told to delay making a prosaccade towards a sudden onset target, saccade latencies were significantly increased compared to when told to make a prosaccade to the target as quickly and as accurately as possible (standard instructions). Under inaccuracy instructions, participants were required to look as fast, but as inaccurately as possible to the target, and these instructions resulted in slower latencies and reduced spatial accuracy compared to standard instructions. Perhaps surprisingly, there appear to have been no systematic investigations into the impact of top-down control on antisaccade performance. Given that the level of top down control is likely to be greater in antisaccades than prosaccade performance, any effects might be expected to be greater on antisaccades compared to prosaccades.

The sudden appearance of the target in the antisaccade task is generally assumed to trigger a motor program for a prosaccade in its direction. According to some accounts, antisaccade errors are to occur when certain endogenous processes fail to inhibit or cancel this program (e.g. Hallet & Adams, 1980). This was articulated most clearly by Everling & Fischer (1998) who argued successful antisaccade performance requires two intact sub-processes, the ability to suppress a reflexive prosaccade towards

the target, and the ability to generate a voluntary saccade in the opposite direction. More recent "parallel programming" accounts of antisaccade performance have suggested that at target onset, a "competitive race" ensues between an exogenously driven prosaccade towards the target and the endogenously driven antisaccade to the opposite side (Massen, 2004; Munoz & Everling, 2004; Reuter & Kathmann, 2004; Reuter, Rakusan, & Kathmann, 2005). These accounts make strong predictions concerning the relationship between correct antisaccade latency and antisaccade error rate, the faster a correct antisaccade can be programmed, the more likely it is to win the race, and be initiated before the incorrect prosaccade towards the target.

In the first study, we sought to establish the extent to which varying task instructions influences antisaccade performance in a sample of healthy participants, and whether any effects on antisaccade error rate are predicted by current competitive race models of antisaccade performance. Standard pro and antisaccade instructions (to the extent that they exist) typically emphasize both speed and accuracy in responding, but it is well known that in other tasks participants may trade one of these aspects of task performance off against the other, for example slowing down responding in order to increase accuracy (e.g. Wickelgren, 1977). We investigated antisaccade performance under instructions that emphasized either speed or accuracy in responding, and also adopted a "delay" instruction as used by Mosimann et al. (2004). Under delay instructions, participants were told to withhold making a response until they were absolutely sure that they had noticed the target's location in peripheral vision, and only then initiate a saccade towards the target on prosaccade trials or its mirror image location on antisaccade trials. We predicted correct antisaccade latencies would be decreased under speed instructions and increased under delay instructions. An additional prediction was antisaccade errors would be reduced under delay instructions.

Experiment 1

Method

Participants

Participants comprised 38 students from the University of Sussex, of whom 14 were male and 24 were female. Ages ranged from 18-47 (M = 27.06, SD = 5.96). All

participants provided written consent and were naïve to the purpose of the study. Three participants' data were excluded, as they did not have a sufficient number of trials, resulting in a final N of 35. A small monetary reward was given upon completion. The study was approved by the departmental ethics committee.

Apparatus

Eye movements were recorded with an Eyelink II eye tracker (SR-Research Ltd., Ontario), with a spatial resolution of 0.25 degrees and a temporal resolution of 2ms. Participants were seated on a modified office chair that prevented any rotational movement, 70cm from the computer screen. Stimuli were displayed on a 21 inch CRT monitor at a refresh rate of 100Hz and a resolution of 1280x1024 pixels. A brief 9 point calibration was done prior to the experiment and repeated if necessary between blocks. Each trial was preceded by a brief drift-correction procedure.

Stimuli

On each trial, the display comprised a black background, containing one red circle (0.25 degrees in diameter), located in the centre of the screen. After a random interval (between 800 and 1200msec), this central stimulus disappeared from the screen and, following a 200msec gap, was replaced by a target stimulus (also a red circle with the same diameter as the central stimulus) which appeared at one of four positions on the horizontal axis (+/- 7.5, or 15degs). The target stimulus was displayed for a variable time between 1500 – 2750msec, which was enough time for a participant to initiate a saccade towards or away from it.

Design/procedures

In a within-participants design, participants performed 256 antisaccade trials, divided into 4 blocks of 64 trials. Each block of trials was performed under one of four different instruction conditions: Standard, Accuracy, Speed and Delay. Participants completed the standard condition first, followed by the three remaining task instructions in counterbalanced order. The standard condition was always performed first as it was important that we established a baseline level of standard antisaccade performance against which to compare the effects of the other instructions. Pilot testing revealed that once some participants had performed a block of trials under the "delay" instructions,

they reported carrying this strategy over into the standard block, despite instructions not to. Testing lasted roughly 15 minutes.

Standard instructions

Participants were told to look to the mirror image location of the re-located target circle as quickly and as accurately as possible.

Accuracy instructions

Participants were told to concentrate on making their saccades as spatially accurate as possible, and emphasised the importance of saccading to the precise mirror image location of the target. Participants were told that speed was not important for these trials, and that they should take as long as they wanted in order to be accurate.

Speed instructions

Participants were instructed to move their eyes as quickly as possible following the target onset to the mirror image location of the re-located target circle. Participants were told that spatial accuracy was not at all important, and it did not matter where their eyes went as long as they went in the opposite direction to the target.

Delay instructions

Participants were told to delay the onset of their saccade until after the target onset. Encouragement was given to only initiate a saccade when they were absolutely positive that they had identified the precise location of the target.

Measures/analysis

Our primary measures of interest were correct antisaccade latency and percentage of antisaccade errors. In addition, we measured final eye position which was defined as the longest stable period of fixation after any corrective saccades had been made. Trials were excluded from analysis if 1) no saccade was made within the trial duration, 2) blinks obscured the primary saccade, 3) the eye was not within 40 pixels, (approximately 1 degree of visual angle) of the central fixation point at the time of target appearance, 4) a saccade was made within 80ms of peripheral target appearance, as these were deemed as anticipations (Wenban-Smith & Findlay, 1991) and 5) the primary saccade was made more than 1000msec after target onset. This resulted in the

exclusion of 6% of trials. Repeated measures ANOVAs were performed on the dependent variables to determine whether any overall effect of condition existed. Huynh-Feldt corrected degrees of freedom are reported when appropriate. Where significant, main effects were followed up with planned comparisons (paired t-tests). Target location was included as a factor in initial analysis but there was no effect of location and no interaction between location and task instructions, so this factor is not presented in the results section.

Results

Correct antisaccade latencies

Correct antisaccade latencies as a function of task instructions are displayed in figure 3.1. A one way repeated measures analysis of variance revealed a significant main effect of task instructions on correct antisaccade latencies ($F(1.89, 64.13) = 45.01$, $p < .001$, $r = .64$, Huynh-Feldt, $e = .78$). Paired t-tests revealed participants were significantly slower to initiate antisaccades under the delay instructions compared to all other instructions ($ts < -8.39$, $ps < .001$, $rs > .65$). When asked to make correct antisaccades as quickly as possible, participants were able to reduce correct latencies by an average of 21msec compared to the standard instructions ($t(34) = 3.48$, $p < .01$, $r = .51$). The accuracy instructions on the other hand, resulted in a significant increase in correct latencies of 52msec compared to the standard instructions ($t(34) = -3.86$, $p < .01$, $r = .55$).

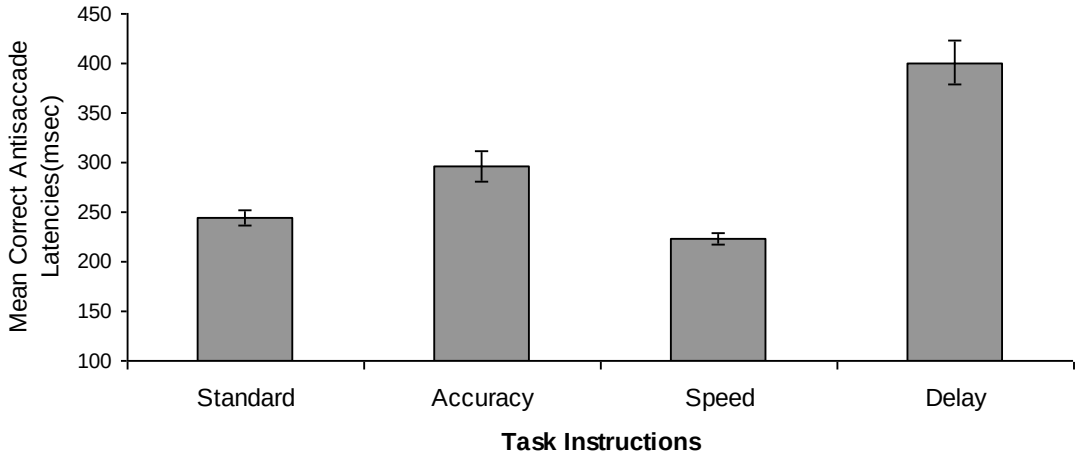

Figure 3.1. Correct antisaccade latencies across different task instructions

Percentage of antisaccade errors

Antisaccade error rate as a function of task instructions is displayed in figure 3.2. A one way repeated measures analysis of variance revealed a significant main effect of task instructions on antisaccade error rate ($F(2.43, 82.66) = 5.42$, $p < .01$, $r = .25$, Huynh-Feldt correction $e = .81$). Paired t-tests showed there was no difference between antisaccade errors in the standard compared to accuracy condition, ($t(34) = -.36$, $p = .72$, $r = .06$). There was also no difference in error rate between the standard and speed conditions ($t(34) = 2.01$, $p = .31$, $r = .32$). However, participants made significantly less errors in the delay condition compared to the standard condition, ($t(34) = 4$, $p < .01$, $r = .57$) and the accuracy condition ($t(34) = 3.78$, $p < .01$, $r = .54$), but not the speed condition ($t(34) = 1.61$, $p = .69$, $r = .27$).

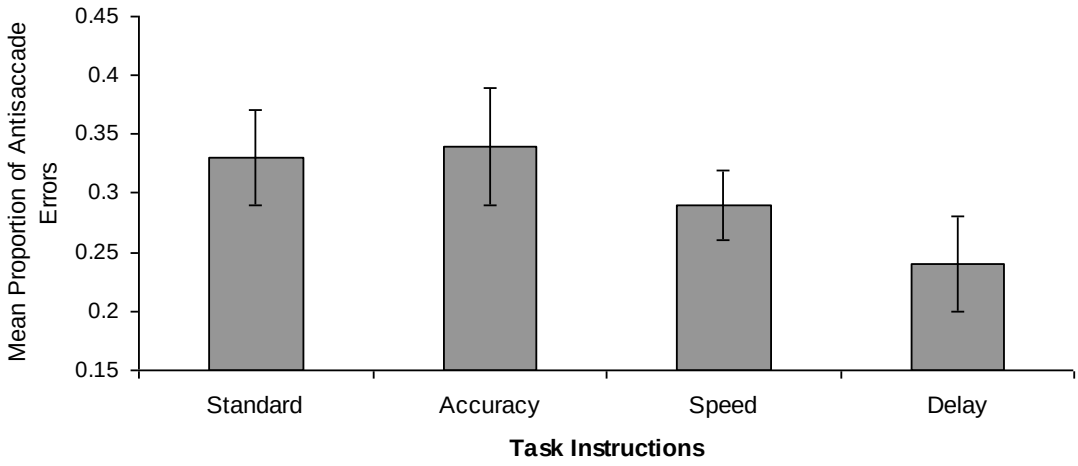

Figure 3.2. Antisaccade error rate across different task instructions

Final eye position

In order to measure spatial accuracy, we calculated final eye position (FEP) (figure 3.3). Final eye position was measured as a ratio, like gain, so an FEP of 1 would be perfect accuracty, whereas a FEP of 1.1 would be a 10% overshoot, and an FEP of .9 would be a 10% undershoot. A one way repeated measures analysis of variance revealed a significant main effect of task instructions on final eye position ($F(2.40, 76.93) = 5.51$, $p < .01$, $r = .26$, Huynh-Feldt, $e = .80$). Paired t-tests revealed that final eye position was more accurate in the accuracy condition compared to the standard condition, ($t(34) = 3.63$, $p = .02$, $r = .53$). Similarly, final eye position was

more accurate under delay instructions compared to standard instructions ($t(34) = 4.08$, $p < .01$, $r = .57$). There was no difference between the standard condition and speed condition ($t(34) = .27$, $p = .79$, $r = .05$). These results suggest that accuracy and delay instructions reduced the overshoot observed under standard instructions.

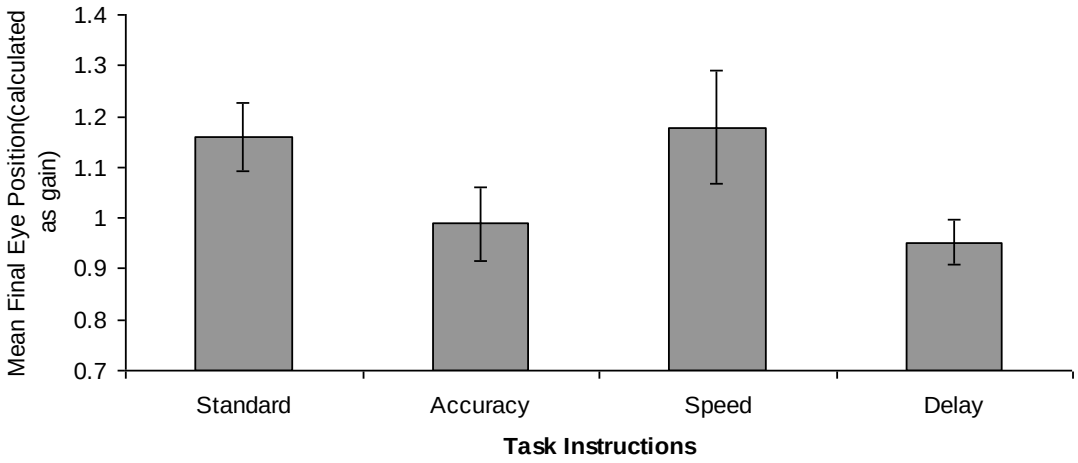

Figure 3.3. Final eye position across different task instructions

Discussion

In the first experiment, we explored the effects of varying task instructions on antisaccade performance. Asking participants to delay their response resulted in increased correct latencies and reduced errors (erroneous prosaccades towards the target) compared to standard instructions. When asked to make an antisaccade as quickly as possible, latencies were reduced, with no significant change in error rate. In contrast when participants were told to be as spatially accurate as possible, correct latencies were increased and again errors were unchanged. Participants were the most spatially accurate under accuracy instructions.

The effects of the speed and accuracy instructions on correct antisaccade latencies suggest that the antisaccade task is vulnerable to speed / accuracy trades offs, in other words one factor influencing the (often large) individual differences in correct antisaccade latency may be participants' personal preference for emphasising either speed over accuracy. If speed were being traded off against accuracy, then instructions to focus on speed would have resulted in a decrease in correct latencies *and* a

subsequent reduction in spatial accuracy, whilst the instructions to focus on accuracy would have resulted in an increase in correct latencies *and* an increase in spatial accuracy. Our results do not suggest a speed/accuracy trade off because accuracy instructions increased latencies and spatial accuracy (final eye position), but speed instructions only reduced latencies, not spatial accuracy. It is possible that spatial accuracy was improved (reduced overshoot in standard condition) under accuracy instructions because it was emphasised, as participants were told to focus on getting the antisaccade as spatially accurate as possible. Similarly, it is possible that spatial accuracy was not altered under speed instructions because it was not emphasised. Furthermore, although spatial accuracy was not emphasised under delay instructions, participants may have shown improved spatial accuracy because of the cautious and 'speed free' strategy they adopted.

Importantly, whilst both the speed and accuracy instructions resulted in significant changes in correct antisaccade latency, neither had any impact on error rate. According to competitive race accounts (Massen, 2004; Munoz & Everling, 2004; Reuter & Kathmann, 2004; Reuter et al., 2005) correct antisaccade latency and antisaccade error rate should be linked, as making faster correct antisaccades should result from activity in the antisaccade pathway reaching threshold first on a greater number of occasions. Similarly, the longer it takes to initiate a correct antisaccade, the more likely it is that activity in the prosaccade pathway would reach threshold first, resulting in an antisaccade error.

In the present experiment, the only condition that affected error rate was the delay condition; when instructed to delay making a response, participants made significantly fewer errors than in any of the other conditions. The effects of the delay instructions on error rate and correct antisaccade latencies also appear to contradict competitive race accounts of antisaccade performance. Unsurprisingly, given the instructions, the delay condition resulted in a marked increase in correct antisaccade latencies. This issue is returned to in the general discussion. The results of the delay condition are important, because they demonstrate that when prompted, participants can readily adopt a simple strategy that can reduce error rates considerably compared to baseline performance. These results suggest that one factor behind the enormous individual differences commonly observed in antisaccade error rate may be the extent to which participants spontaneously adopt a similar "delay response" strategy.

It is difficult to explain why there was no change in error rate for both accuracy and speed conditions, despite an increase and a decrease in correct latencies respectively. One possibility is that increases or decreases in correct antisaccade latency need to be above a certain threshold before any impact on error rate is observed. In order to further test the extent to which these findings challenge competitive race accounts of antisaccade performance, the effects of these same task instructions on prosaccade performance need to be established. According to race accounts (e.g. Massen, 2004) if a manipulation has similar effects on correct prosaccade and antisaccade latencies, then there should be no impact on antisaccade error rate, as the likelihood of either the prosaccade or antisaccade program reaching threshold first remains unchanged. Therefore in our second study we included both prosaccade and antisaccade trials to test this.

Experiment 2

In the second study, we sought to establish the extent to which varying task instructions influences both pro and antisaccade performance in a sample of healthy participants. Race model accounts suggest any manipulation that affects correct prosaccade and antisaccade latencies to the same degree will not impact on antisaccade error rates (Massen, 2004). We tested this by including both prosaccade and antisaccade trials. The instructions used were identical to the first experiment. On the basis of the first study, we predicted that correct antisaccade latencies would be significantly reduced under delay instructions. A further prediction was that correct prosaccade latencies would be reduced under delay instructions and in line with the race model, it was expected that no change in antisaccade error rate would occur, as prosaccade and antisaccade latencies would be affected to the same degree.

Method

Participants

Participants comprised 28 students of whom 8 were male and 20 were female. Ages ranged from 19-30 (M = 21.35, SD = 3.10). All participants provided written consent and were naïve to the purpose of the study. Data from one participant was not

included in the final analysis as they did not have a sufficient number of trials. Another participant was removed, as they failed to understand experimental instructions in one condition, resulting in a final N of 26. The study was approved by the departmental ethics committee.

Apparatus

These were identical to experiment 1.

Stimuli

This was identical to the previous experiment, except now, the target stimulus could appear in one of six locations on the horizontal axis, (+/- 5, 10, or 15degs).

Design/procedures

In a within-participants design, participants performed a total of 576 trials, 288 prosaccade and 288 antisaccade trials, each divided into 4 blocks of 72 trials. Prosaccade trials were performed first. As we were interested in the impact of task instructions on antisaccade error rate, we felt it important to maximise the potential number of errors. Performing a large number of prosaccade trials serves to firmly establish a "prosaccade" set in the experimental situation, thus potentially increasing the number of errors in the antisaccade blocks. Conditions were identical to the first study, as was order. For both prosaccade and antisaccade tasks participants completed the standard condition first, followed by the three remaining task instructions in a counterbalanced order (accuracy, speed, delay). Testing lasted roughly 40 minutes.

Standard instructions

For prosaccade trials participants were instructed to look at the peripheral target as quickly and as accurately as possible. For antisaccade trials, participants were told to look to the mirror image location of the re-located target circle as quickly and as accurately as possible.

Accuracy instructions

For prosaccade trials participants were told to concentrate on making their saccades as spatially accurate as possible, and looking precisely at the centre of the target when it appeared. Participants were told that speed was not important for these

trials, and that they should take as long as they wanted in order to be accurate. For antisaccade trials, the instructions were the same, but emphasised the importance of saccading to the precise mirror image location of the target.

Speed instructions

For both prosaccade and antisaccade trials participants were instructed to move their eyes as quickly as possible following the target onset (to the target in prosaccade trials and to the mirror image location in antisaccade trials). Participants were told that spatial accuracy was not at all important, and it did not matter where their eyes went as long as they went towards the target (or away from it on antisaccade trials) as quickly as possible.

Delay instructions

In both pro and antisaccade trials participants were instructed to delay the onset of their saccade until after the target onset. To achieve this they were encouraged to only initiate a saccade (either towards the target on prosaccade trials or to the mirror image location on antisaccade trials) when they were absolutely positive that they had identified the precise location of the target.

Measures/analysis

Our primary measures of interest were correct prosaccade and antisaccade latency and percentage of antisaccade errors. Trial exclusion was identical to the first study. This resulted in the exclusion of 6% of trials. Repeated measures ANOVAs were performed on the dependent variables to determine whether any overall effect of condition existed. Huynh-Feldt corrected degrees of freedom are reported when appropriate. Where significant, main effects were followed up with planned comparisons (paired t-tests).

Results

Correct prosaccade and antisaccade latencies

Correct prosaccade and antisaccade latencies as a function of task instructions are displayed in figure 3.4. A 2 (task: prosaccade vs. antisaccade) by 4 (instruction: standard, accuracy, speed, delay) repeated measures ANOVA revealed a significant

main effect of task ($F(1, 25) = 130.71$, $p < .001$, $r = .92$). Overall correct antisaccade latencies were significantly slower than correct prosaccade latencies. The main effect of instructions was also significant ($F(1.18, 29.59) = 47.88$, $p < .001$, $r = .79$, Huynh-Feldt, $e = .39$) but was qualified by a significant instructions by task interaction $F(1.52, 38.02) = 8.21$, $p < .01$, $r = .42$ (Huynh-Feldt correction $e = .51$). As a result, separate one way ANOVAs were performed on the prosaccade and antisaccade data.

For the prosaccade data a main effect of instructions ($F(1.06, 26.54) = 42.49$, $p < .001$, $r = .78$, Huynh Feldt, $e = .35$), was followed up with paired t-tests that revealed participants were significantly slower to initiate prosaccades under the delay instructions compared to all other instructions (all $ts < -6.67$, $ps < .001$, $rs > .79$). Asking participants to make prosaccades as quickly as possible did not result in a decrease in correct latencies ($t(25) = .1.1$, $p = .28$, $r = .21$) and asking participants to focus instead on accuracy did not result in an increase in latencies compared to standard instructions ($t(25) = -1.80$, $p = .08$, $r = .34$).

A main effect of instructions was also significant for correct antisaccade latencies ($F(1.38, 34.43) = 38.95$, $p < .001$, $r = .73$, Huynh-Feldt, $e = .46$). As with prosaccade trials, the "delay" instructions resulted in an increase in correct antisaccade latencies (of ~160msec) compared to the other three conditions (all $ts < -7.3$, $ps < .001$ $rs > .75$). In contrast with their effect on prosaccades, the instructions to focus on either speed or accuracy had marked impacts on antisaccade performance. When asked to make correct antisaccades as quickly as possible participants were able to reduce correct antisaccade latencies by an average of 26msec compared to standard instructions ($t(25) = 4.98$, $p < .001$, $r = .71$). The accuracy instructions, on the other hand, resulted in a significant increase ($t(25) = -3.85$, $p = .001$, $r = .61$) in correct antisaccade latencies of 48msec compared to the standard instructions.

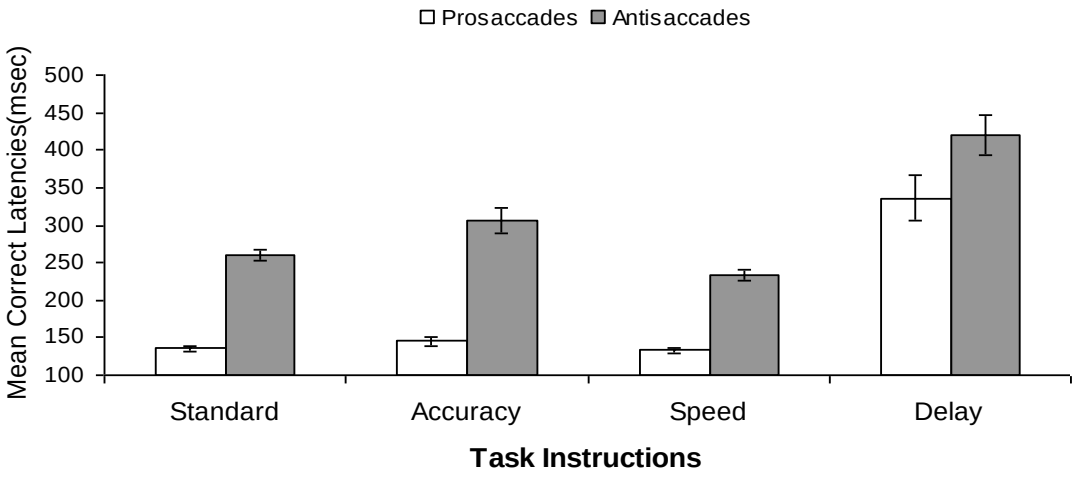

Figure 3.4. Correct latencies across different task instructions as a function of saccade task

Percentage of antisaccade errors

Percentage of antisaccade errors are plotted as a function of task instructions in figure 3.5. A one way repeated measures analysis of variance revealed a main effect of task instructions ($F(2.56, 63.96) = 11.52$, $p < .001$, $r = .39$, Huynh-Feldt correction, $e = .65$). Paired t-tests indicated that there was no difference between antisaccade errors in the standard compared to accuracy condition, ($t(25) = .10$, *ns*, $r = .02$). There was a trend towards participants making more errors under speed instructions compared to standard instructions ($t(25) = -1.89$, $p = .07$, $r = .35$). Participants made significantly less errors in the delay condition compared to the standard condition, ($t(25) = 3.35$, $p < .01$, $r = .56$) and the speed and accuracy conditions (both $ts > 3.77$, $ps < .001$, $rs > .60$).

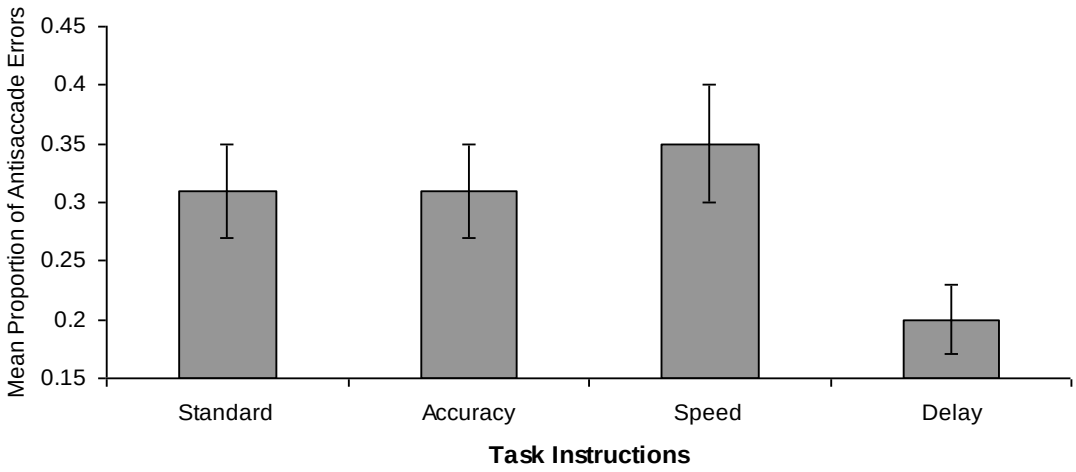

Figure 3.5. Antisaccade error rate across different task instructions

Correlations

In order to explore the relationship between correct latency and antisaccade error rate as a function of task instruction, we performed a series of correlations. Under standard instructions the correlation between prosaccade latency and antisaccade error rate failed to reach significance (r = -.32, p = .11). However, the correlation was significant under accuracy instructions (r = -.42, $p < .05$) and approached significance under speed instructions (r = -.37, p = .06). Under delay instructions the correlation was highly significant (r = -.58, $p < .002$).

Discussion

In the second experiment, we explored the effects of varying task instructions on both pro and antisaccade performance. Asking participants to delay making a saccade to the target (prosaccade trials), or to its mirror image location (antisaccade trials) resulted in increased latencies compared to standard trials. As in experiment 1, antisaccade error rate was reduced under delay instructions compared to all other instructions. When asked to make a saccade as quickly as possible, correct latencies were not significantly reduced for prosaccades, but were for antisaccades with no significant change in antisaccade error rate (again replicating experiment 1). Latencies were unchanged under accuracy instructions for prosaccade trials, but were significantly reduced for antisaccade trials, with no change to antisaccade error rate (again replicating experiment 1). Interestingly, correct prosaccade latencies were a strong predictor of antisaccade error rate for delay instructions.

The effect of delay instructions on prosaccade latencies replicates previous work by Mosimann et al. (2004) who also found that participants delayed the onset of prosaccades by around 200msec when instructed. The fact that participants could delay making an antisaccade is less surprising than their ability to delay making a prosaccade to the target. Asking someone to delay making an eye movement is something that requires a volitional act from the participant. Therefore, one would expect this manipulation to be more applicable to antisaccade performance as the antisaccade task relies on more top-down processes, whereas the prosaccade task is more concerned with bottom-up processes. The present results imply that participants are able to suppress the

tendency to saccade to the target and use top-down processing to volitionally delay making a prosaccade.

It is likely that the speed and accuracy instructions had little impact on correct prosaccade latencies due to the relative ease of the prosaccade task. A manipulation that requires a participant to be as fast as possible (such as speed instructions) may be comparatively ineffective in the prosaccade task, as latencies under standard instructions are already fast in the first place. Similarly, a manipulation that focuses on accuracy may not affect prosaccade latencies; because participants would not have to slow down to ensure accuracy as much on prosaccade trials, as they would on antisaccade trials.

Consistent with our previous study, antisaccade error rate was significantly reduced under delay instructions compared to all other conditions. According to the race model (Massen 2004), antisaccade error rate should not be affected if a manipulation affects pro and antisaccade latencies to the same degree. It is difficult to accommodate the present findings into race accounts because our results show that the delay manipulation influenced pro and antisaccade latencies to the same degree. In other words, activity in the separate prosaccade (exogenous) and antisaccade (endogenous) pathways were both influenced, which should not have given any advantage to the antisaccade pathway. One potential explanation of this finding, which we explore further in experiment 3 and in the general discussion, is provided by Reuter, Jager, Bottlender, & Kathmann (2007). They suggest an alternative explanation to current race model accounts of antisaccade performance when trying to explain delay antisaccade conditions. They argue that a competition arises between prosaccade activity and fixation activity. If fixation activity is greater than prosaccade activity, then lower error rates should occur. Therefore it would appear that in the present experiment, instructions to delay making an antisaccade have encouraged greater levels of fixation activity resulting in fewer antisaccade errors. In other words, only activity in fixation neurons was influenced by the manipulation which gave an advantage to fixation activity over prosaccade activity.

Previous studies have documented correlations between prosaccade latency and antisaccade error rate in healthy participants (e.g. Roberts, Hager, & Heron, 1994; Taylor & Hutton, 2007). It is not clear why we failed to observe this relationship in the standard instruction condition in the present experiment, although as the correlation was in the expected direction, it may simply be a matter of power. Interestingly, the

correlation was strongest under delay instructions as participants who delayed their prosaccades the least were those who made most errors in the delayed antisaccade task. In general these findings support race models in that participants with the fastest prosaccade latencies were those with the highest error rates. The fact that this relationship was strongest in the condition that explicitly required participants to attempt to delay their responding suggests that the extent to which participants can exert top down control over saccade initiation is a critical component of the relationship between prosaccade latency and antisaccade error rate.

Given that all external task parameters (e.g. timings/size and locations of target) remained identical across the four conditions, the marked reduction in antisaccade error rate that occurred when participants were instructed to delay responding compared to standard instructions demonstrates the sensitivity of the task to changes in task parameters that are entirely internally generated. It also suggests the possibility that individual differences in the strategic approach participants adopt when performing the antisaccade task may be a significant factor in explaining the high variability in error rate typically observed (e.g. Evdokimidis et al., 2002).

Experiment 3

The delay instructions used in experiments 1 and 2 resulted in marked increases in both pro and antisaccade correct latencies and a reduction in antisaccade error rate. One potential explanation of these results is that the delay instructions served to increase activity in the fixation neurons of the superior colliculus and participants were able to maintain fixation in a central position, despite the offset of the central stimulus and the onset of the peripheral target. In this respect, the delay instructions seem to result in behaviour equivalent to that observed in "delayed" pro and antisaccade tasks that have been used in the psychiatric literature (e.g. Reuter et al., 2005; Reuter et al., 2007). In a standard delayed prosaccade task, participants are asked to maintain central fixation after a peripheral target has appeared until a tone or other "go-signal" is given, usually 500-1000msec later. The task thus shares with the antisaccade task the potential for participants to make stimulus driven erroneous prosaccades towards the target. Previous research suggests that such errors are typically reduced in delayed prosaccade tasks compared to standard antisaccade tasks (Hutton, Joyce, Barnes, & Kennard 2002;

Reuter et al., 2005, 2007). Interestingly, errors in a delayed antisaccade task (in which participants are instructed to delay the antisaccade until a go signal) are also reduced compared to a standard antisaccade task. Reuter et al. (2007) suggest that under delayed instructions fixation activity is relatively higher at the point of target onset compared to standard instructions, and due to the inhibitory pathways between collicular fixation neurons and movement neurons, an erroneous prosaccade towards the target is less likely to be initiated.

In contrast with the standard delayed prosaccade tasks described above, in experiments 1 and 2 the central fixation point was removed prior to the target's appearance, and was absent for the remainder of the trial. In other words, fixation was driven endogenously (by the intention to delay the saccade) as opposed to exogenously (by a central stimulus) during the delay period. Also the delay instructions did not specifically emphasise the need to maintain fixation, and participants may have differed in the extent to which they chose to delay initiating a saccade. In order to standardise the requirement to maintain fixation across participants in experiment 3 we compared standard prosaccade and antisaccade performance with "delayed" versions of these tasks, in which participants were instructed not to make prosaccades or antisaccades until a tone signal had been delivered at some point after the target onset. In addition, we included a "fixation" task in which we removed the requirement for participants to make a saccade when the target appeared. This task served as a control condition, which allowed us to establish the extent to which explicit instructions to maintain fixation served to inhibit prosaccades towards sudden onset targets. In all tasks, the central fixation point was removed 200msec prior to the target appearing. We predicted that participants will make fewest errors in the fixation task, and fewer errors in the delayed pro and antisaccade task compared to the standard antisaccade task.

Method

Participants

Participants comprised 25 students of whom 7 were male and 18 were female. Ages ranged from 19-30 (M = 21.35, SD = 3.10). All participants provided consent and were naïve to the purpose of the study. Data from one participant was excluded, as they did not have a sufficient number of correct trials to provide meaningful estimates of

correct antisaccade latency, resulting in a final N of 24. The study was approved by the departmental ethics committee.

Apparatus

The apparatus used was identical to the previous experiments.

Stimuli

For all tasks, the stimuli used were identical to experiments 1 and 2. The central red fixation circle disappeared after 800-1200msec, and following a 200msec gap an identical target circle appeared in one of four locations, either +- 7.5, or 15degs from the centre. The target stimulus was displayed for a variable time between 1500 – 2750msec. As there were no instructions in this experiment to increase spatial accuracy, having a large number of potential target locations was not important. The preliminary analyses from experiment 2 revealed no significant location by instruction interaction, so in order to simplify the task, but maintain an element of uncertainty as to target location, the number of locations was reduced from 6 to 4. In the delayed tasks a 200Hz tone that lasted 100msec was delivered 550, 850, 1150 or 1450msec after the target onset. The tone served as a "go signal". The variation in the delay period was introduced in order to minimise anticipatory saccades.

Design and Procedures

In a within-participants design, participants performed 64 trials of 5 different oculomotor tasks: (1) Fixation, (2) prosaccades, (3) delayed prosaccades, (4) antisaccades and (5) delayed antisaccades. The fixation task was always performed first, and the order of the remaining four tasks was counterbalanced across participants. No practice trials were given, and testing lasted around 30 minutes.

Fixation instructions

Fixation trials required participants' to maintain looking at the central fixation point throughout the duration of the whole trial, without making any saccades towards or away from the target stimulus.

Standard prosaccade and antisaccade instructions

For prosaccade trials participants were instructed to look at the peripheral target as quickly and as accurately as possible. In the antisaccade task they were instructed to look to the mirror image location of the peripheral target as quickly and as accurately as possible.

Delayed prosaccade and antisaccade tasks

For prosaccades and antisaccades, participants were instructed to delay the onset of their saccade until they heard the auditory cue. To do this they were encouraged to keep looking at the central stimulus location until they heard a beep sound. Upon hearing this, they were required to saccade to the target (delayed prosaccade task), or to the mirror image location to the target (delayed antisaccade task).

Measures/analysis

We established the percentage of erroneous prosaccades made towards the target on the fixation, antisaccade, delayed prosaccade and delayed antisaccade tasks. A delayed prosaccade error was any saccade made towards or away from the target prior to the tone signal, or any saccade made away from the target after the tone signal. A delayed antisaccade error was any saccade made towards or away from the target prior to the tone signal, or any saccade towards the target after the tone signal. Therefore errors were either 'jump the gun' errors (saccade made before tone signal) or direction errors (saccade made in the wrong direction after tone). In addition, we recorded correct saccade latencies for the prosaccade, antisaccade, delayed prosaccade and delayed antisaccade tasks. Criteria for excluding trials were identical to the first study. In order to discount prosaccade errors on the delayed tasks that occurred as a result of anticipations to the onset of the tone, rather than being triggered by the onset of the target, trials with prosaccade errors with latencies more than 2 times the standard deviation of the participants' prosaccade latencies were removed from the analysis (c.f. Reuter et al., 2005). This resulted in ~5% of delayed prosaccade /antisaccade trials being removed. As the correct response in the fixation task does not result in a saccade, this condition was not included in this analysis.

Results

Correct prosaccade and antisaccade latencies

Correct latencies as a function of task instructions are plotted in figure 3.6. A one way repeated measures analysis of variance revealed a significant main effect of task (F (1.8, 41.32) = 38.15, $p < .001$, $r = .69$, Huynh-Feldt, $e = .65$). Paired t-tests revealed that participants were significantly faster under prosaccade instructions, compared to all other task instructions ($ts < -14.35$, $ps < .001$, $rs > .78$). In addition, correct delayed antisaccades were significantly slower than correct delayed prosaccades ($t(25) = -3.74$, $p < .001$, $r = .36$). The latencies of delayed antisaccades and standard antisaccades did not differ ($t(25) = .56$, $p = .58$, $r = .11$).

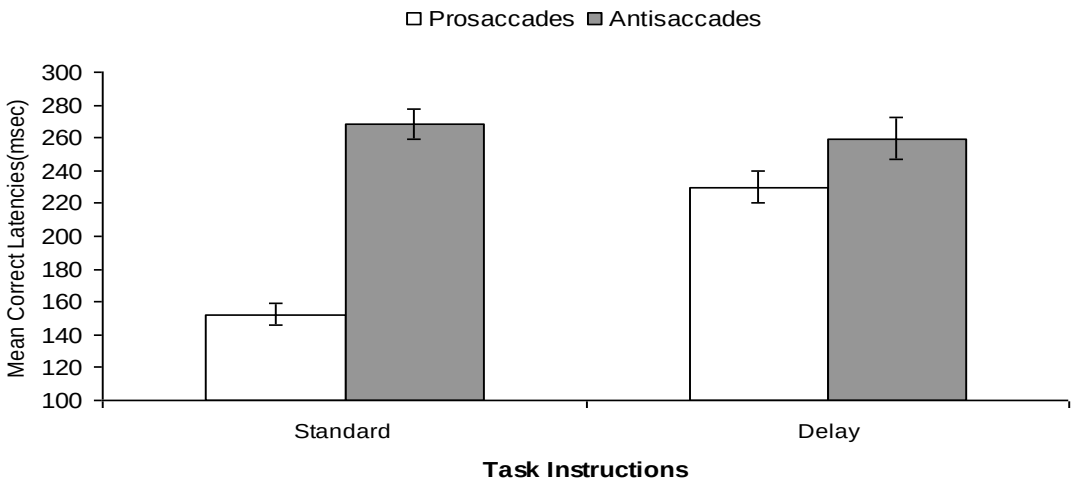

Figure 3.6. Correct latencies across different task instructions as a function of saccade task

Percentage of fixation, prosaccade and antisaccade errors

The error rate analysis was divided into two parts. The first part of the analysis looked at differences in errors between the different task conditions, where delayed prosaccade and delayed antisaccade errors were any saccade made towards or away from the target prior to the tone signal (jump the gun errors). The second part of the analysis looked at differences in errors between the different task conditions, where delayed prosaccade and delayed antisaccade errors were any saccades made in the wrong direction after the tone signal (direction errors).

Percentage of errors as a function of task is plotted in figure 3.7. A one way repeated measures analysis of variance was conducted on fixation, delayed prosaccade (jump the gun errors) antisaccade and delayed antisaccade errors (jump the gun errors). There was an overall main effect of task ($F(1.97, 45.30) = 20.25$, $p < .001$, $r = .56$, Greenhouse Geisser, $e = .66$). Planned comparisons using paired samples t-tests revealed that participants made very few errors on the fixation task compared to any of the other three tasks ($ts < 5.66$, $ps < .002$, $rs > .69$). Interestingly, participants made significantly more errors on the delayed prosaccade task compared to the antisaccade task $t(23) = 2.06$, $p = .05$, $r = .39$. Participants made a similar amount of errors on the delayed prosaccade task compared to the delayed antisaccade task and on the antisaccade task compared to the delayed antisaccade task ($ts < 1.76$, $ps > .05$ $rs < .34$).

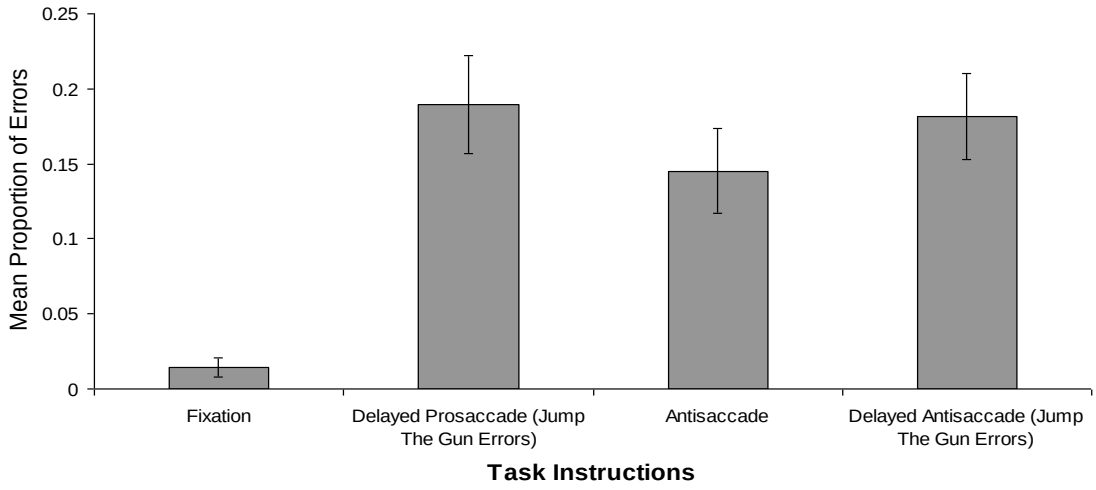

Figure 3.7. Average errors across different task instructions

Percentage of errors as a function of task is plotted in figure 3.8. A one way repeated measures analysis of variance was conducted on fixation, delayed prosaccade (direction errors) antisaccade and delayed antisaccade errors (direction errors). There was an overall main effect of task ($F(1.09, 25.09) = 22.52$, $p < .001$, $r = .69$, Greenhouse Geisser, $e = .36$). Planned comparisons using paired samples t-tests revealed that participants made more errors on the antisaccade task compared to any of the other three tasks ($ts < 4.94$, $ps < .002$, $rs > .69$). There were no other differences in errors between tasks.

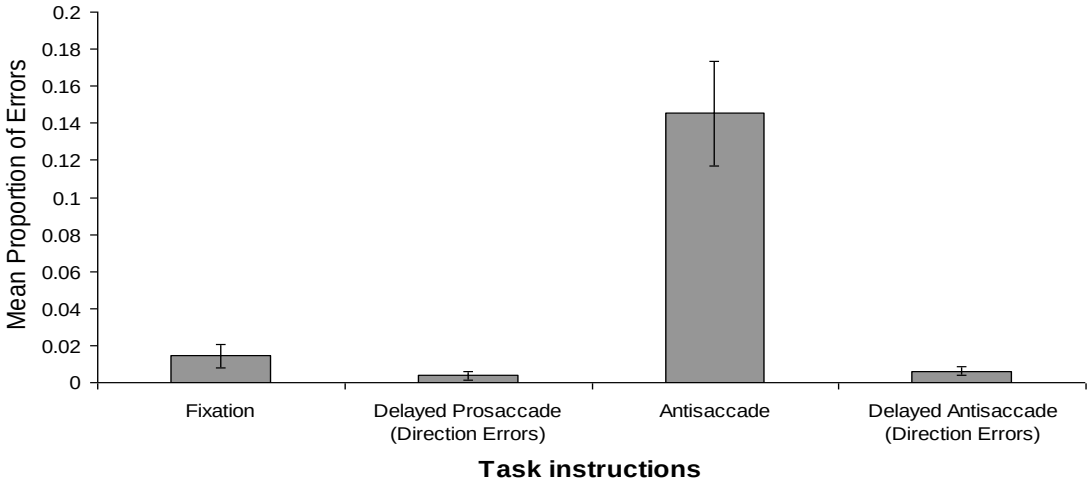

Figure 3.8. Average error rate across different task instructions

Discussion

Participants correct latencies were increased in the delayed prosaccade condition compared to the standard prosaccade condition. However, participants' correct latencies were unchanged in the delayed antisaccade condition compared to the standard antisaccade condition. It took longer for participants to make delayed antisaccades than delayed prosaccades. In terms of error rate, participants made few errors under fixation instructions and very few direction errors under delay prosaccade and antisaccade instructions. Only antisaccade direction errors (not jump the gun errors) were reduced under delay instructions, compared to standard antisaccade instructions.

Under prosaccade instructions, participants' correct latencies were faster than all other instructions. This finding replicates Reuter et al. (2007) who used similar tasks, and is consistent with previous research demonstrating that exogenously driven saccades have faster latencies than endogenously driven saccades (e.g. Walker, Walker, Husain & Kennard, 2000). However, whereas we observed that correct delayed antisaccade latencies were slower than correct delayed prosaccade latencies, Reuter et al. did not find this effect. One key difference between the delayed antisaccade and delayed prosaccade tasks is that a "vector transformation" is required in the antisaccade task in order to provide the co-ordinates of the mirror image location. In our study there were four different possible goal locations, compared to only two in Reuter et al's study and it is possible that with only two locations participants interpret the task as "saccade

left" or "saccade right" and place less emphasis on making spatially accurate saccades (in terms of amplitude).

Under fixation instructions error rate was very low compared to most other tasks and both Hutton et al. (2002) and Reuter et al. (2007) also found that healthy participants made very few errors on similar tasks. This finding supports the suggestion that activity in fixation neurons inhibits activity in collicular movement neurons. This in turn decreases the probability of activity reaching the threshold required for an erroneous prosaccade towards the target to be triggered (Munoz & Fecteau, 2002). Compared to the standard antisaccade task, participants made fewer erroneous saccades towards the target (direction errors) in the delayed pro and antisaccade tasks. Meyniel, Rivaud-Pechoux, Damier, & Gaymard (2005) found that patients with progressive supranuclear palsy and fronto-temporal dementia also made significantly fewer anticipatory prosaccades, compared to healthy controls in a delayed antisaccade task compared to a standard antisaccade task. Similarly, Reuter et al. (2005) found that both healthy participants and patients with schizophrenia made fewer prosaccade errors in a delayed antisaccade task compared to a standard antisaccade task. Moreover, Walker, Husain, Hodgson, Harrison, & Kennard (1998), found that a patient with frontal lobe damage was better able to suppress errors in a fixation task compared to an antisaccade task. Together, these findings support the suggestion that fixation neurons are likely to be more active in these delayed tasks at the time of target onset compared to the standard antisaccade task, and therefore limit the activity of movement neurons, reducing the likelihood of an erroneous prosaccade being made towards the target.

The finding that errors were more likely to occur (although not significantly more likely) in the delayed prosaccade task compared to the delayed antisaccade task is important, as it cannot be explained by differences in baseline levels of activity in fixation neurons, as these should be equivalent in both tasks. In both the delayed prosaccade task and delayed antisaccade task participants are required to refrain from making a saccade (either pro or anti) until the given signal. One possibility (discussed below) is that baseline activity in the neurons that support prosaccades towards targets is higher in the delayed prosaccade task compared to the delayed antisaccade task.

An additional finding was that participants made fewer direction errors compared to 'jump the gun' errors when instructed to delay making a pro or antisaccade. It is not surprising that direction errors were minimal for the delayed prosaccade task, as like the standard prosaccade task, this task only requires you to

saccade in the direction of the target stimulus. However, it is not clear why direction errors in the delay antisaccade task were also low, as attention is more likely to be captured by the onset target, as it is in the standard antisaccade task. One possibility is that delaying the antisaccade encourages greater fixation, which means fixation neurons are better able to inhibit activity in movement neurons, resulting in the reduced probability of activity in the error prosaccade pathway reaching the threshold for saccade triggering. The dfference in jump the gun errors and direction errors under delay instructions suggests that participants were able to use the delay period to increase directional control over their eye movements, but the expectation of the presentation of the tone signal may have facilitated more anticipatory saccades.

General Discussion

Across three experiments, we manipulated task instructions in order to determine their effect on pro and antisaccade performance. We found that requiring participants to delay their response (for an internally generated duration in experiments 1 and 2, and a task imposed duration in experiment 3) resulted in a significant increase in correct antisaccade latency and a significant reduction in antisaccade error rate. Instructions to make antisaccades as quickly as possible resulted in a decrease in correct antisaccade latency, whereas instructions to focus on accuracy increased correct latencies. Neither of these instructions had any effect on antisaccade error rate or prosaccade latencies. Instructions to fixate on the centre of the screen were shown to be successful in inhibiting erroneous prosaccades.

The finding that providing participants with a simple verbal strategy can significantly reduce error rates has important implications. Researchers have had limited success in identifying individual differences that can account for significant proportions of the large variability in antisaccade error rate typically observed in healthy participants. For example, Smyrnis et al. (2003) found that only 1% of the variance in antisaccade performance was explained by differences in schizotypal personality traits in a large sample of healthy participants. Other studies have found only modest associations between antisaccade error rate and measures of working memory function (e.g. Hutton et al., 2004). The present results suggest that one determinant of antisaccade performance may be the strategic approach taken by the participant. Those

participants who adopt a strategy that focuses on maintaining fixation when the target appears are likely to make significantly fewer errors than those who choose to respond as quickly as possible. Patients with schizophrenia demonstrate significantly increased error rates (see Hutton & Ettinger, 2006 for a review), and have also been shown to have deficiencies in adopting successful strategies when performing cognitive tasks (Hutton et al., 1998; Christensen, Girard, Benjamin, & Vidailhet, 2006). It would be interesting to determine the extent to which error rates can be improved in such patients simply by providing them with task instructions that emphasise a delaying strategy.

The finding that delay instructions result in similar increases in prosaccade and antisaccade latencies but result in a marked reduction in antisaccade errors also has implications for current competitive race accounts of antisaccade performance (Massen, 2004; Munoz & Everling, 2004; Reuter & Kathmann, 2004; Reuter et al., 2005). These models draw on "accumulator" models of saccade generation which assume that saccades are triggered when neural activity accumulates from a baseline level to the point at which it passes a certain threshold (Carpenter 1981; Carpenter & Williams 1995; Hanes & Schall, 1996; Trappenberg, Dorris, Munoz, & Klein, 2001). The time taken for the activity to reach the threshold (i.e. the latency of the saccade) can vary as a function of the baseline level of activity, the rate of rise in the activity, or the level at which the threshold is set.

Drawing on these models and evidence that saccades can be programmed in parallel (e.g. Godijn & Theeuwes, 2002; Walker & McSorley, 2006), Massen (2004) argues that antisaccade errors occur when activity in the neural systems supporting the exogenously triggered prosaccade reach threshold before activity in the neural systems supporting the endogenous antisaccade. As a result, an erroneous prosaccade is made towards the target, followed rapidly by the correct antisaccade when activity in its neural systems reaches the threshold for saccade triggering. In correct trials, activity supporting the antisaccade reaches threshold first, thus "winning" the competition and the build up of activity supporting the erroneous prosaccade towards the target ceases. Competitive race accounts predict that any manipulation that differentially affects prosaccade and antisaccade latencies will consequently influence the error rate, as it will influence the likelihood of one of the processes reaching threshold first. In support of this prediction, Massen (2004) showed that manipulations that result in increased latencies for correct antisaccades (such as reducing the probability of antisaccade trials in a block of mixed prosaccade and antisaccades) also result in increased errors.

Therefore if the endogenous antisaccade is slower, there is a greater probability of the exogenously triggered prosaccade winning the race and reaching the threshold for activation first.

According to Massen (2004) a manipulation that results in significantly increased correct antisaccade latencies (such as the delay instructions used in experiments 1 & 2) should either result in an increase in antisaccade error rate, or no change in error rate if the manipulation increases prosaccade latencies to a similar degree. We found that error rates were in fact markedly reduced when participants were instructed to delay their saccades. Reuter et al. (2007) explain a similar result (reduced antisaccade errors in a delayed antisaccade task) by suggesting that in conditions that encourage fixation at the time of target onset, the competition is not necessarily between competing exogenously and endogenously driven saccades, but between fixation and the exogenously driven saccade. According to Reuter et al. (2007) errors are reduced in such situations as fixation provides a more efficient source of competition for the erroneous prosaccade, reducing baseline activity sufficiently that the likelihood of activity surpassing threshold after target onset is reduced. Neurophysiological studies provide support for this suggestion. The rostral pole of the superior colliculus (SC) contains cells that are active during fixation, whereas more caudal neurons in the SC are topographically organised neurons whose activity directs the eyes to another position in space (Munoz & Istvan, 1998; Munoz & Wurtz, 1993). It has been suggested that fixation and saccade related neurons in the SC may actually exist on a continuum with "fixation" neurons coding for much smaller movements than the saccade related neurons (Krauzlis, Basso, & Wurtz, 1997). Whatever their relationship, these cells appear to operate reciprocally (Everling, Dorris, Klein, & Munoz, 1999) as the more active fixation neurons are, the less active saccade neurons are (Munoz & Fecteau, 2002).

Thus, a modified version of competitive race models, in which competition can exist between neural systems supporting fixation and those supporting the correct response, as well as competition between neural systems supporting the incorrect prosaccade and correct antisaccade could explain the reduction in antisaccade errors observed under delayed instructions in experiments 1 & 2. In experiment 3 however, we found that error rate was greater (all be it not significantly) in the delayed prosaccade condition compared to the delayed antisaccade condition. In both tasks activity in collicular fixation neurons should be equivalent, as both required the same delay before

the initiation of a response. One possible explanation of this result, that is compatible with competitive race models, is that it reflects differences in the baseline activity in saccade related neurons. Activity in the neurons supporting a prosaccade towards the target is likely to have a higher baseline level of activity in the delayed prosaccade task compared to the delayed antisaccade task as the delayed prosaccade task requires a prosaccade response to be made. In other words, when participants are expecting to make a prosaccade, this expectation is reflected in increased activity in neurons supporting the prosaccade. Increased activity in prosaccade neurons increases the probability of an erroneous prosaccade being made towards the target.

Under "speed" instructions in experiments 1 & 2, participants were able to produce faster correct antisaccades than under standard instructions. The instructions to make saccades as quickly as possible had no impact on prosaccade latencies however. According to race model accounts of antisaccade performance, if participants are able to generate faster correct antisaccade responses, the likelihood of activity in the prosaccade pathway reaching threshold first should be reduced. As a result, faster correct antisaccade responding should lead to a reduction in antisaccade error rate. This was not found. Instead, error rate increased marginally, as would be predicted if participants were trading speed off against accuracy. Similarly, instructions to make spatially accurate antisaccades led to an increase in correct antisaccade latency, but no subsequent increase in antisaccade error rate, as would be predicted by competitive race models. It is possible that increases or decreases in correct antisaccade latency need to be above a certain threshold before any impact on error rate is observed. The increase in antisaccade error rate under speed instructions was not statistically significant (although the effect size was moderate), and further experiments will be required in order to establish the conditions under which manipulations that result in increases in correct antisaccade latencies lead to reductions or increases in error rate.

In conclusion, antisaccade performance has been shown to be highly sensitive to differences in task instructions. By extending competitive race models to allow competition to exist between neural activity supporting fixation and the erroneous prosaccade towards the target, the main finding that delayed instructions resulted in reduced errors despite increasing correct antisaccade latencies can be accommodated. Further research is required in order to determine the circumstances under which manipulations that result in faster correct antisaccade latencies also result in fewer errors.

Article 4 - The Role of Top-down Factors on Antisaccade Error Awareness

A revised and abbreviated version of this paper has been accepted in: *Experimental Brain Research.*

Abstract

Antisaccade errors occur when participants saccade towards a sudden onset target, despite having been told to ignore it and look to the opposite side. One relatively unexplored feature of antisaccade performance is that participants are unaware of a large proportion of the errors they make (Mokler & Fischer, 1999). Across two experiments we explored the extent to which antisaccade error awareness is altered by manipulations known to affect antisaccade metrics such as error rate and correct latency. In experiment 1, participants performed the antisaccade task under standard instructions, instructions to respond as quickly as possible or instructions to delay responding. Instructions to 'delay' making an antisaccade significantly reduced antisaccade error rate compared to 'standard' and 'speed' instructions. This reduction was driven by a decrease in the number of errors that participants were aware of task instructions did not alter the number of "unaware" errors. In experiment 2, participants performed antisaccades only, antisaccades with spatial tapping (ST) and antisaccades with a random number generation task (RNG). The number of "aware" antisaccade errors was increased in the two dual task conditions compared to the single task condition, but unaware error rates remained unchanged. These results are discussed in light of recent models of antisaccade performance.

Introduction

The antisaccade task requires participants to initiate a saccade to the mirror image location of a sudden onset target, and is widely used as a convenient tool for studying the cognitive processes associated with goal-directed behaviour. On around 20% of trials, healthy participants erroneously saccade towards the sudden onset target, before making a correct antisaccade to the mirror image location (Smyrnis et al., 2002). It is generally assumed that all antisaccade errors are in some sense "equal" and reflect a failure to inhibit a prosaccade towards the target, or sufficiently activate the intention to initiate an antisaccade (e.g. Everling & Fischer, 1998; Findlay & Walker, 1999; Rycroft, Hutton & Rusted, 2006). There is, however, some evidence to suggest that different "types" of antisaccade error may be usefully distinguished. Mokler & Fischer (1999), for example, found that healthy participants are typically only aware of around 50% of the antisaccade errors they make. The time taken for participants to correct "aware errors" was significantly longer than the time they took to correct errors of which they were unaware. The authors proposed that unaware errors may take less time to correct because in these instances attention was not directed towards the sudden onset target (despite the fact that a saccade is triggered towards it).

Nieuwenhuis, Ridderinkhof, Blom, Band, & Kok (2001) also explored error awareness in antisaccade performance. Participants were presented with a white central fixation circle and four surrounding yellow squares, to the left, right, above and below centre. After a brief interval, the outline of one square thickened (pre-cue) and following this, a cue stimulus (white circle) was presented inside one of the squares. Finally, a target stimulus (cross) was presented, always in the square opposite to where the cue had been presented. On each trial, participants were instructed to move their eyes to the target cross location (i.e. make an antisaccade to the opposite side of the cue). Participants were instructed to press the space bar if they thought they had moved their eyes in the direction of the cue, thus indicating a perceived error. Electroencephalography (EEG) recordings were taken during task performance. The EEG data revealed that the event related potential 'error positivity', or (Pe) was more pronounced for aware errors compared to unaware errors. The Pe is a slow positive potential with centroparietal distribution, which often follows an 'error negativity potential' or (Ne) on incorrect trials and usually occurs 300-500msec, 80msec after a

response (Fiehler, Ullsperger, & Cramon, 2005). In support of Mokler & Fischer (1999) participants were unaware of a large proportion of their antisaccade errors (50±25%) and the erroneous saccades that participants were not aware of had shorter correction times and smaller amplitudes compared to aware errors. The results of these studies suggest several differences in antisaccade metrics between aware and unaware antisaccade errors.

Other research has confirmed that aware and unaware errors are associated with different electrophysiological signals. Fiehler et al. (2005) found differences in levels of event related negativity (ERN) for different types of corrected errors using a flanker task. Participants were presented with a fixation mark for about 500msec at the centre of a screen, after which four flanker arrows appeared, above and below the centre of the screen for 110msec. A target arrow was also presented with the flanker arrows. In congruent trials the flanker arrows pointed in the same direction as the target arrow, whereas in incongruent trials the flanker and target arrows pointed in different directions. Participants had to respond using their left hand if the target arrow pointed to the left and with their right hand if the target pointed to the right. Half of their participants were instructed to immediately correct any errors they made (intentional error correction) and the other half were unaware that corrective responses were recorded (incidental error correction). The authors reported differences in levels of event related negativity (ERN) for intentional and incidental error correction. In addition, they found that an early peak of ERN was associated with errors that were corrected rapidly and a late peak of ERN was associated with errors that were corrected more slowly.

Together, these findings suggest that aware and unaware antisaccade errors are dissociable both in terms of their properties (amplitudes and correction times) and neural signatures, raising the possibility that they may also respond differentially to experimental manipulations that have previously been shown to impact on antisaccade performance. Specifically, the small amplitudes, rapid correction time and reduced Pe amplitude associated with unaware errors suggest that they may not necessarily arise from the same failure of inhibition (or goal activation) that is generally assumed to underlie antisaccade errors.

One high level manipulation that has been shown to have powerful impact on antisaccade performance is task instruction set. Taylor & Hutton (2009) gave healthy participants separate blocks of antisaccades with different verbal instructions. Under

standard instructions, participants were asked make an antisaccade as quickly and as accurately as possible. Under accuracy instructions, participants were told to focus on ensuring spatial accuracy and to take as much time as required to make a spatially accurate response. With speed instructions, participants were asked to disregard spatial accuracy and attempt to initiate a correct antisaccade as fast as they possibly could. Finally, for delay instructions, participants were asked to delay making an antisaccade until they were absolutely certain they had identified the target. Compared to standard instructions, antisaccade errors were reduced under delay instructions. In addition, correct antisaccade latencies were decreased under speed instructions.

The aim of experiment 1 was to systematically explore the role of different task instructions on error awareness in antisaccade performance, to establish whether these instructions differentially effect aware vs. unaware errors. We predicted that task instructions will impact on the proportion of aware antisaccade errors that participants make, but not on the number of unaware errors because it is assumed that only aware errors arise from the same failure of goal activation that is associated with antisaccade errors. Specifically, based on the error results from our previous study (Taylor & Hutton, 2009), we expected aware errors would be reduced under 'delay' instructions and increased under 'speed' instructions, compared to 'standard' instructions.

Experiment 1

Method

Participants

Participants comprised 44 students (30 females) from the University of Sussex aged 19 – 40 (M = 23.36, SD = 5). Participants were paid a small monetary reward for taking part. Data from 2 participants was excluded, as they did not have enough valid trials, after all invalid trials had been excluded resulting in a final N of 42. The study was approved by the departmental ethics committee.

Apparatus

Eye movements were recorded using an Eyelink II eye tracker (SR-Research Ltd., Ontario, Canada) with a temporal resolution of 2ms and a spatial resolution of

around 0.25 degs. The stimuli were displayed on a 21inch CRT monitor with a screen resolution of 1280 x1024 pixels and a refresh rate of 100Hz. Actual screen dimensions were 40cm horizontal and 30cm vertical. Participants were seated approximately 60cm from the screen in an adjustable chair that had been modified to prevent any rotational movement. Each set of trials was preceded by a calibration procedure, during which participants focused their eye gaze on 3 separate targets on the horizontal axis. Only right eye movements were recorded.

Stimuli

On each trial, the display comprised a black background, containing one red circle (0.25 degrees in diameter), located in the centre of the screen. After a random interval (between 800-1200msec), this central stimulus disappeared from the screen and, following a 200msec gap, was replaced by a target stimulus (also a red circle with the same diameter as the central stimulus) which appeared at one of four positions on the horizontal axis, (+/- 7.5, or 15 degs). The target stimulus was displayed for a variable time between 1500 – 2750msec which was enough time for a participant to initiate a saccade towards or away from it.

Design/procedures

In a within-participants design, participants performed 216 antisaccade trials, divided into 3 blocks of 72 trials. Each block of trials was performed under one of 3 different task instructions: Standard, Speed and Delay (see paper 3 for description) in a fully counterbalanced order. At the end of each trial, participants stated whether they made a correct antisaccade by saying ‘yes’ or an error by saying ‘no’. This was recorded by the experimenter. There were no practice trials and testing lasted around 25 minutes.

Standard instructions

Participants were told to look to the mirror image location of the re-located target circle as quickly and as accurately as possible.

Speed instructions

Participants were instructed to move their eyes as quickly as possible following the target onset to the mirror image location of the re-located target circle. Participants

were told that spatial accuracy was not at all important, and it did not matter where their eyes went as long as they went in the opposite direction to the target.

Delay instructions

Participants were told to delay the onset of their saccade until after the target onset. Encouragement was given to only initiate a saccade when they were absolutely positive that they had identified the precise location of the target.

Measures/analysis

Our primary measures of interest were correct antisaccade latency and percentage of antisaccade errors. We also measured the time it took participants to correct an error (antisaccade error correction time) and antisaccade amplitude. Amplitude was denoted by 'gain' which was calculated as the ratio of the actual saccade amplitude divided by the desired saccade amplitude. Trials were excluded from analysis if 1) no saccade was made within the trial duration, 2) blinks obscured the primary saccade, 3) the eye was not within 40 pixels, (approximately 1 degree of visual angle) of the central fixation point at the time of target appearance, 4) a saccade was made within 80ms of peripheral target appearance, as these were deemed as anticipations (Wenban-Smith & Findlay, 1991) and 5) the primary saccade was made more than 1000msec after target onset. This resulted in the exclusion of 8% of trials. Repeated measures ANOVAs were conducted on the data to attempt to find main effects of condition and interactions between the factors. Paired t-tests were used to compare conditions.

Results

Correct antisaccade latencies

Correct antisaccade latencies as a function of task instructions are plotted in figure 4.1. A one way repeated measures ANOVA revealed a significant main effect of task instructions on correct antisaccade latencies ($F(1.38, 56.66) = 59.78$, $p < .001$, $r = .72$, Greenhouse-Geisser, $e = .69$). Paired t-tests showed that participants were significantly faster under standard instructions compared to delay instructions ($t(41) = 7.91$, $p < .001$, $r = .78$). Similarly, participants were faster to make a correct antisaccade when given speed instructions compared to delay instructions ($t(41) = 8.42$, $p < .001$, r

= .80). However, the difference in correct antisaccade latencies was not significant between the standard condition and the speed condition ($t(41) = 1.75$, $p = .26$, $r = .26$).

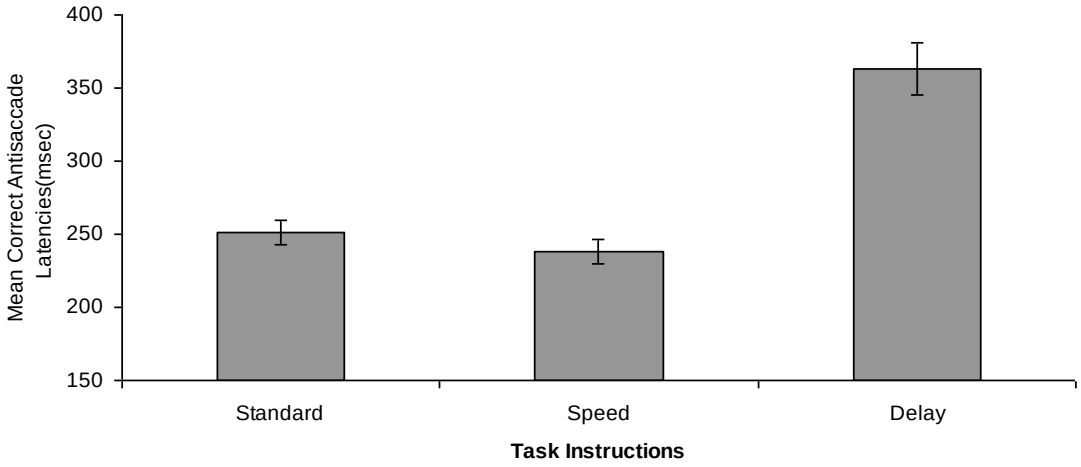

Figure 4.1. Correct antisaccade latencies across different task instructions

Percentage of antisaccade errors

Percentage of antisaccade errors as a function of condition is plotted in figure 4.2. A one way repeated measures ANOVA revealed a significant effect of condition ($F(1.77, 72.74) = 8.84$, $p < .001$, $r = .33$, Huynh-Feldt, $e = .89$). Paired t-tests found that participants made significantly fewer antisaccade errors under delay instructions compared to standard instructions ($t(41) = 2.89$, $p < .001$, $r = .41$). Similarly, participants made fewer antisaccade errors under delay instructions compared to speed instructions ($t(41) = 3.52$, $p = .001$, $r = .48$). There was a trend for participants to make more errors under the speed instructions compared to standard instructions ($t(41) = 1.82$, $p = .08$, $r = .27$).

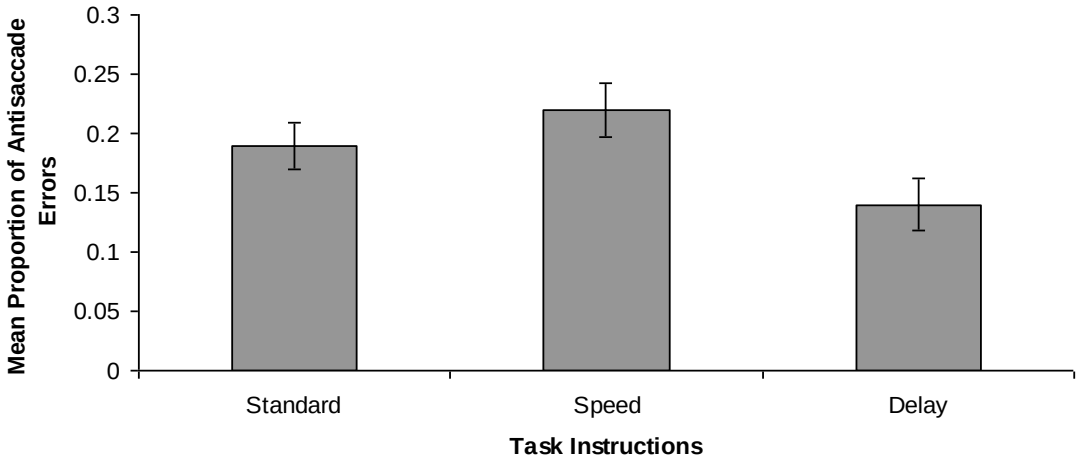

Figure 4.2. Antisaccade error rate across different task instructions

Percentage of aware and unaware antisaccade errors

Percentage of antisaccade errors across different task instructions as a function of awareness is plotted in figure 4.3 A two way repeated measures ANOVA with task instructions and awareness as factors revealed a significant main effect of task instructions on antisaccade errors ($F(1.77, 72.74) = 8.84$, $p = .001$, $r = .33$, Huynh-Feldt, $e = .89$). There was also a main effect of awareness on antisaccade errors, as participants were aware of a higher percentage of errors than they were unaware ($F(1, 41) = 10.37$, $p < .01$, $r = .45$). The interaction between instructions and awareness was also significant ($F(1.67, 68.51) = 15.34$, $p < .001$, $r = .43$, Huynh-Feldt, $e = .84$). The interaction occurred because unaware error rate was similar across all task instructions, but a dramatic drop was found for aware errors in the delay condition, compared to the standard and speed conditions.

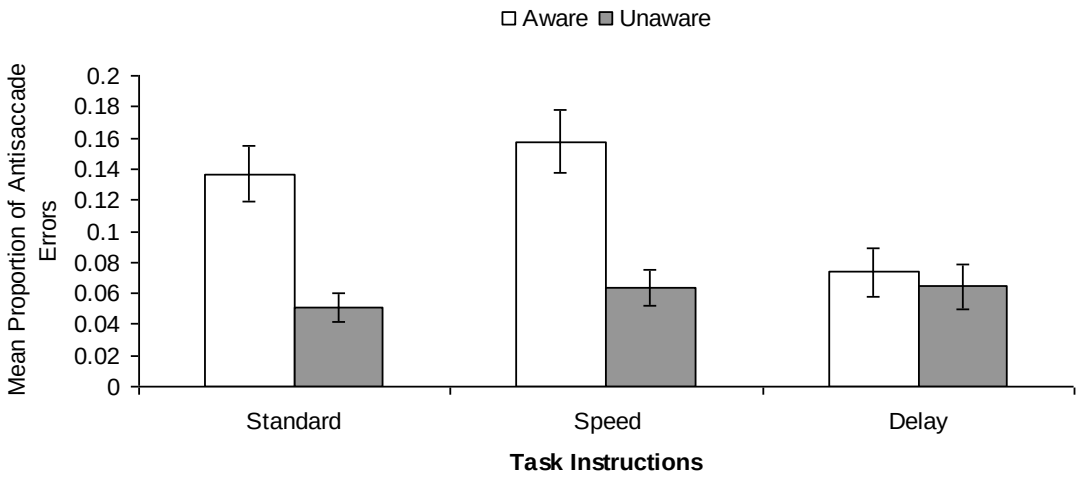

Figure 4.3. Antisaccade error rate across different task instructions as a function of awareness

This impression was confirmed with separate one way ANOVAs on the aware and unaware data. For the aware data, there was a main effect of task instructions on aware antisaccade errors ($F(2, 82) = 19.53$, $p < .001$, $r = .44$). Paired t-tests showed that delay instructions reduced aware errors compared to standard instructions ($t(41) = 5.52$, $p < .001$, $r = .65$). Similarly, delay instructions reduced aware antisaccade errors compared to speed instructions ($t(41) = 5.74$, $p < .001$, $r = .67$). However, there was no difference in the amount of aware errors made between standard and speed instructions ($t(41) = 1.34$, $p = .56$, $r = .20$). For the unaware data, there was no main effect of task instructions ($F(1.53, 62.83) = .75$, $p = .44$, $r = .11$, Greenhouse-Geisser, $e = .77$).

Antisaccade amplitudes

On average, participants saccadic amplitudes for 'aware' antisaccade errors (M = .81 SD = .13, range = .46-1.00), were significantly larger than participants saccadic amplitudes for 'unaware' antisaccade errors (M = .66, SD = .16, range = .09 - .90, ($t(37) = 7.15$, $p < .001$, $r = .76$).

Antisaccade error correction times

On average, participants were significantly slower to correct 'aware' antisaccade errors (M = 249.65msec, SD = 116.21, range = 122.19msec – 772msec), compared to 'unaware' antisaccade errors (M = 175.44msec, SD = 60.63, range = 90msec – 322msec, ($t(37) = 3.80$, $p = .001$, $r = .53$).

Discussion

Instructions to delay making antisaccades resulted in an increase in correct antisaccade latencies and a reduction in error rate, whereas instructions to respond as quickly as possible led to a slight increase in error rate (significant at trend level). These findings were consistent with our previous study (Taylor & Hutton, 2009). Overall, participants were generally aware of more of their antisaccade errors than they were unaware. Critically, the delay instructions altered the proportion of aware antisaccade errors, but not the proportion of unaware errors. Unaware antisaccade errors had smaller amplitudes and corrected much faster compared to aware antisaccade errors. These findings supported previous research (Mokler & Fischer, 1999).

The fact that 'delay' instructions reduced aware error rates, but not unaware error rate provides further evidence that these two types of errors may have different underlying mechanisms. This apparent distinction between aware and unaware errors is also supported by the results from the speed condition, as aware errors were increased (allbeit not significantly) and unaware errors remained unaffected under speed instructions. In addition, the difference in saccade amplitudes and correction times of errors when comparing aware and unaware errors further suggests that different types of antisaccade errors can occur.

The 'delay' instructions are believed to reduce antisaccade error rates due to an increase in 'fixation activity.' According to Reuter, Jager, Bottlender, & Kathmann (2007), in delay antisaccade conditions, a competition arises between fixation neuron activity and prosaccade neuron activity and if fixation activity is greater than prosaccade activity, then lower error rates should occur. In the present study, it is possible that only activity in fixation neurons were influenced by the delay manipulation which gave an advantage to fixation activity over prosaccade activity, resulting in a decrease to antisaccade errors compared to standard instructions. Similarly, this explanation may also account for the decrease in 'aware' antisaccade errors under delay instructions. This finding supports the suggestion that activity in fixation neurons inhibits activity in collicular movement neurons, which in turn reduces the probability of activity in the prosaccade pathway reaching the threshold for triggering a saccade (Munoz & Fecteau, 2002). However, this explanation does not account for unaware antisaccade errors under delay instructions. The fact that unaware errors were largely unaffected by delay

instructions suggests that prosaccade activity remained relatively high during unaware errors resulting in an increased chance of antisaccade errors.

In experiment 2 we sought to determine whether a manipulation that results in an increase in antisaccade errors also has its impact through aware errors alone. Previous research has shown that performing a secondary task that makes demands on working memory processes can significantly increase the number of antisaccade errors made (e.g. Mitchell, Macrae, & Gilchrist 2002; Roberts, Hager & Heron, 1994). We used two different secondary tasks, one that was designed to specifically reduce central executive resources (a random number generation task) and the other designed to tax spatial working memory (a spatial tapping task). We predicted that antisaccade errors would be increased by both secondary tasks, and on the basis of the results of experiment 1, we predicted that the secondary tasks would increase the number of aware errors that participants made, but not the number of unaware errors.

Experiment 2

Method

Participants

Participants comprised 21 students (16 females) from the University of Sussex aged 19 – 35 (M = 23.7, SD = 3.98). Participants had normal to corrected vision and received course credits for taking part. The study was approved by the departmental ethics committee. One participant was excluded on the grounds that the majority of their trials were excluded.

Apparatus/stimuli

Apparatus used and the stimuli presented were identical to experiment 1, except that a metronome was used to keep the spatial tapping in time.

Design/procedures

In a within-participants design, participants performed 192 antisaccade trials, divided into 3 blocks of 64 trials. In a fully counterbalanced order, participants completed antisaccades only, antisaccades with spatial tapping and antisaccades with

random number generation. At the end of each trial, participants stated whether they thought they had made a correct antisaccade by saying ‘yes’ or an error by saying ‘no’. This was recorded by the experimenter on paper. There were no practice trials and testing lasted around 30 minutes.

Antisaccades only

In this condition, participants had to complete a block of standard antisaccades.

Antisaccades with spatial tapping (ST)

Participants had to simultaneously perform a block of antisaccades whilst tapping numbers 1-9 on the keyboard in order, one key per second. An electronic metronome was used to keep participants in time. Participants were able to practise the finger tapping at the beginning of the experiment.

Antisaccades with random number generation (RNG)

In this condition, participants had to simultaneously perform a block of antisaccades whilst generating a random sequence of the digits 1-9, at a rate of one digit per second. Again, a metronome was used to keep participants in time.

Measures/Analysis

The measures and analysis were identical to experiment 1.

Results

Correct antisaccade latencies

Correct antisaccade latencies as a function of condition are plotted in figure 4.4. A one way repeated measures ANOVA revealed a significant main effect of condition ($F(2, 38) = 10.05$, $p < .001$, $r = .45$). Paired t-tests showed that participants were significantly faster to make correct antisaccades when performing antisaccades alone, compared to antisaccades with spatial tapping (ST) ($t(20) = 4.02$, $p < .01$, $r = .68$). In addition, participants were also faster to make a correct antisaccade when performing antisaccades with random number generation (RNG) compared to antisaccades with ST ($t(19) = 3.01$, $p = .02$, $r = .57$). However, there was no difference in correct antisaccade

latencies when participants performed antisaccades alone compared to antisaccades with RNG ($t(19) = .82$, $p = 1$, $r = .18$).

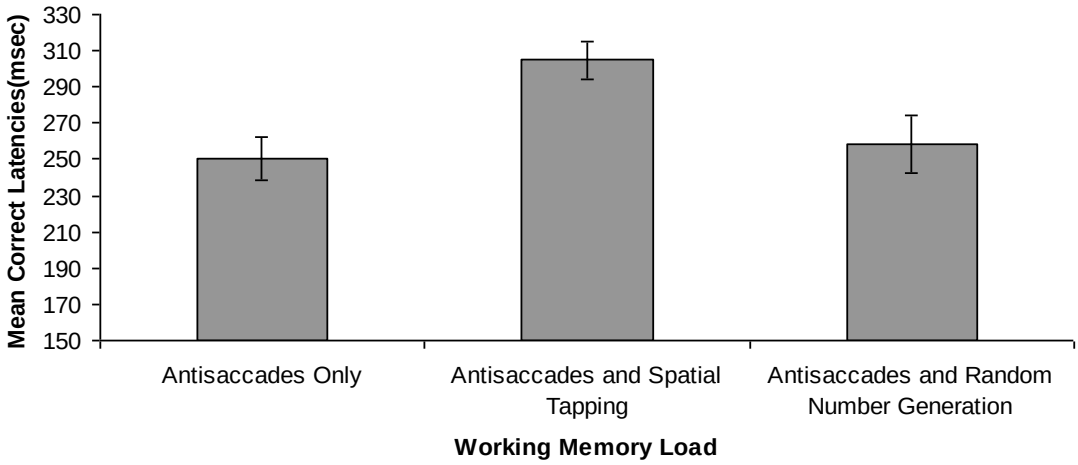

Figure 4.4. Correct antisaccade latencies across working memory load

Percentage of antisaccade errors

Percentage of antisaccade errors as a function of condition is plotted in figure 4.5. A one way repeated measures ANOVA revealed a significant main effect of condition ($F(2, 38) = 9.86$, $p < .001$, $r = .45$). Paired t-tests showed that participants made fewer antisaccade errors when performing antisaccades only compared to antisaccades with ST ($t(19) = 3.57$, $p < .01$, $r = .63$). Similarly, participants made fewer antisaccade errors when performing antisaccades only compared to antisaccades with RNG ($t(19) = 3.41$, $p < .01$, $r = .62$). However, participants made a similar amount of errors when performing antisaccades with ST and antisaccades with RNG ($t(19) = 1.42$, $p > .05$, $r = .31$).

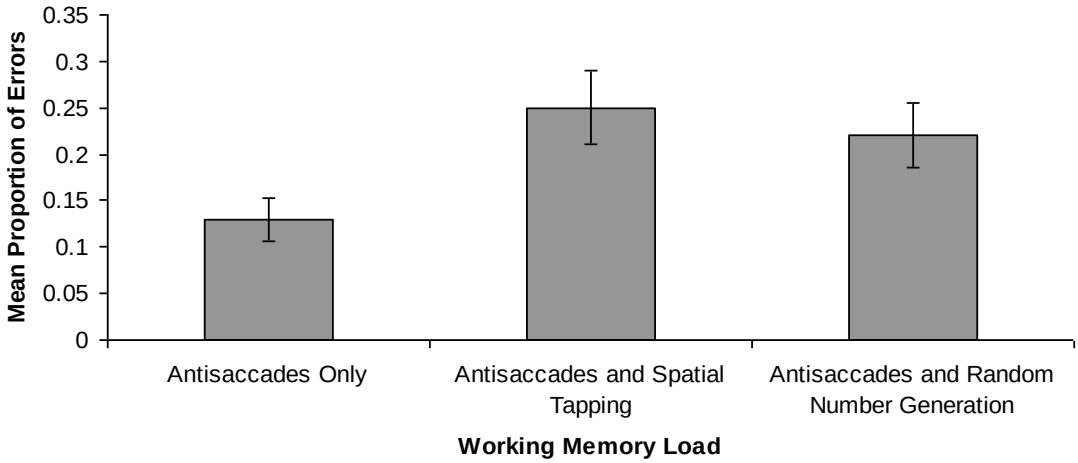

Figure 4.5. Antisaccade error rate across working memory load

Percentage of aware and unaware antisaccade errors

Percentage of antisaccade errors across different working memory loads as a function of awareness is plotted in figure 4.6. A two way repeated measures ANOVA with working memory load and awareness as factors revealed a significant main effect of working memory load on antisaccade error rates ($F(2, 38) = 9.86$, $p < .001$, $r = .45$). There was also a main effect of awareness, as participants were aware of more of their errors than they were unaware ($F(1, 19) = 7.89$, $p = .01$, $r = .54$). The interaction between working memory load and awareness was also significant ($F(2, 38) = 3.22$, $p = .05$, $r = .28$). The interaction occurred because participants were generally more aware of their antisaccade errors when performing an additional working memory task, but neither more aware or unaware of errors when performing antisaccades only (see figure 4.6).

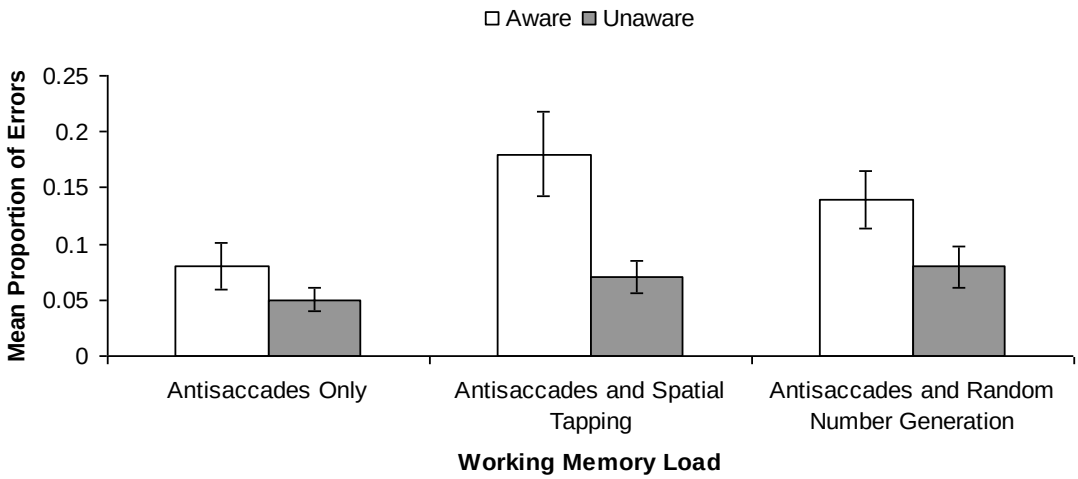

Figure 4.6. Antisaccade error rate across working memory load as a function of awareness

The two way interaction was explored with separate one way ANOVAs on the aware and unaware data. For the aware data, there was a main effect of working memory load on antisaccade errors ($F(1.27, 24.03) = 7.43$, $p < .01$, $r = .49$, Greenhouse-Geisser, $e = .63$). Paired t-tests showed that participants' proportion of aware antisaccade errors was higher in the antisaccades with ST condition, compared to the antisaccades only condition ($t(19) = 2.89$, $p = .02$, $r = .55$). Similarly, participants' proportion of aware errors was higher in the antisaccades with RNG condition compared to the antisaccades only condition ($t(19) = 2.75$, $p = .03$, $r = .53$). However, there was no difference in the proportion of aware errors made in the antisaccades with ST condition compared to the antisaccades with RNG condition ($t(19) = 2.09$, $p > .05$, $r = .43$).

For the unaware data, there was no main effect of working memory load on antisaccade errors ($F(1.40, 26.64) = 1.67$, $p > .05$, $r = .24$, Greenhouse-Geisser, $e = .70$).

Antisaccade amplitudes

On average, participant's saccadic amplitudes for 'aware' antisaccade errors ($M = .80$ $SD = .12$, range = .58 – .99), were significantly larger than participant's saccadic amplitudes for 'unaware' antisaccade errors ($M = .67$, $SD = .20$, range = .21 - .1.0, ($t(19) = 2.66$, $p = .01$, $r = .52$).

Antisaccade error correction times

On average, participants were significantly slower to correct 'aware' antisaccade errors (M = 251.81msec, SD = 72.10, range = 105msec – 363.63msec), compared to 'unaware' antisaccade errors (M = 185.40msec, SD = 59.62, range = 72msec – 309.33msec, ($t(18) = 2.84$, $p = .01$, $r = .56$).

Discussion

Antisaccade error rates increased when participants performed antisaccades with spatial tapping (ST) and antisaccades with random number generation (RNG) compared to antisaccades alone. However, both secondary tasks increased the aware error rate but not the unaware error rate. As in experiment 1, the proportion of errors which participants were unaware of remained unaffected by the manipulation. In support of experiment 1, saccade amplitudes were larger and correction times of errors took longer for aware antisaccade errors compared to unaware errors.

The finding that performing a secondary task increased antisaccade error rate is consistent with previous studies (e.g. Mitchell et al., 2002; Roberts et al., 1994). Both the secondary tasks used in the present study were designed to tax specific working memory processes. Previous work has suggested that the random number generation task (RNG) is particularly efficient at reducing executive processes, particularly those involved in inhibition, updating, and monitoring (Peters, Giesbrecht, Jelicic, & Merckelbach, 2007). These executive processes are believed to play a role in antisaccade performance as well (e.g. Everling & Fischer, 1998).

The spatial tapping (ST) task used in the present study was intended to tax visuospatial sketchpad resources, a component of Baddley's working memory model associated with spatial working memory (Baddeley, 2000). Past research has suggested that spatial working memory is particularly important for antisaccade performance (e.g. Hutton et al., 2004; Niemann et al., 2000).

Interestingly, in the present study, only the ST task resulted in an increase in correct antisaccade latencies. This may have occurred simply because of the difference in complexity of the two tasks. However, previous dual task studies have found mixed results regarding correct antisaccade latencies. Roberts et al. (1994) found that correct latencies were increased when participants' performed antisaccades and mental

arithmetic compared to antisaccades alone. In contrast, Mitchell et al. (2002) found no difference in correct latencies when participants performed antisaccades and the n-back task compared to antisaccades alone.

The fact that ST and RNG affected aware error rate, but not unaware error rate, supports the results from experiment 1 and confirms the apparent distinction between these two types of antisaccade errors in healthy participants. In addition, the difference in saccade amplitudes and error correction times between aware and unaware errors supports this distinction. The results from experiment 2 suggest that a top-down manipulation capable of increasing antisaccade errors has also impacted on aware but not unaware errors. In the general discussion that follows, these findings are considered in light of recent models of antisaccade performance.

General discussion

Across two experiments we explored the impact of top-down factors on antisaccade error awareness. In experiment 1, we manipulated the task instructions given to participants. We found that compared to standard instructions, 'delay' instructions decreased antisaccade errors and increased correct antisaccade latencies, a direct replication of our previous research (Taylor & Hutton, 2009). Importantly, task instructions and awareness interacted, the delay instructions reduced the number of errors that participants were aware of, but had no impact on the number of unaware errors which remained constant at around 6%. In experiment 2 we found that performing a secondary task increased the number of antisaccade errors that participants were aware of, but again, the number of unaware errors was unaffected, and remained at around 5%. In both studies, participants' saccades were larger and error times were longer for aware errors compared to unaware errors.

These findings support previous research that "aware" and "unaware" antisaccade errors differ in a number of respects (Mokler & Fischer 1999; Nieuwenhuis et al., 2001). They showed that errors of which participants were aware had greater amplitudes and took longer to correct than unaware errors. Mokler & Fischer (1999) speculated that the increased correction time for aware errors occurs because in these instances, attention is first directed towards the sudden onset target, before being redirected to the correct mirror image location, a time consuming process, whereas in

unaware errors, attention is not directed towards the sudden onset target (even though a saccade has been). The findings here extend Mokler & Fischer's results, and provide further support for the hypothesis that the two types of errors may have different underlying mechanisms by demonstrating that the two types of error are differentially sensitive to experimental manipulation.

Within this model, it would appear that in the present study "delay" instructions decreased the likelihood that attention was allocated to the target, whereas dual task manipulations increased it. According to Reuter et al. (2007), in delay antisaccade paradigms, prosaccade activity and fixation activity compete. If the build up of fixation activity is greater than the build up of prosaccade activity at the point of target onset, then this should reduce the chance of an erroneous prosaccade reaching the threshold for triggering a saccade first. This explanation may also account for why participants' proportion of 'aware' antisaccade errors was reduced under delay instructions. In line with this explanation, it is possible that during a delayed antisaccade which a participant is aware of, activity in the prosaccade pathway is reduced as a result of increased fixation activity. In other words, activity in fixation neurons inhibits activity in collicular movement neurons, which in turn reduces the probability of activity in the prosaccade pathway reaching the threshold for triggering a saccade (Munoz & Fecteau, 2002). As unaware antisaccade errors were unaffected by delay instructions, it is assumed that fixation activity did not exceed prosaccade activity on unaware trials, and the probability of activity in the prosaccade pathway reaching the threshold for triggering a saccade remained high.

Goal activation accounts of antisaccade performance have argued in favour of a strong relationship between working memory and antisaccade performance (Eenshuistra, Ridderinkhof, & Van der Molen, 2004; Nieuwenhuis, Broerse, Nielen, & de Jong 2004). These accounts suggest that antisaccade errors are most likely to occur when there is insufficient goal activation within the working memory system. Specifically, if the appropriate 'antisaccade task set' (Fischer & Weber, 1996) is activated, then the likelihood of making a correct antisaccade is improved. Moreover, Nieuwenhuis et al. (2004) suggested that goal activation levels can be mediated by instructions, environmental structure, and concurrent task requirements. In experiment 1 of the present study, delay instructions have clearly mediated levels of goal activation, as a manipulation capable of reducing attention towards the onset target (such as delay

instructions), can enable participants to better access the 'antisaccade task set' and reduce errors.

In experiment 2, the added requirement of completing a secondary task whilst performing antisaccades increased antisaccade error rate. According to Roberts et al. (1994), antisaccade error rate is increased in these 'dual task' paradigms because the secondary task (such as the RNG or ST used in the current study) reduces working memory resources needed to make an antisaccade. Therefore the addition of a secondary task will decrease goal activation and increase the likelihood of an antisaccade error being made. However, the relationship between goal activation and awareness of antisaccade error rate remains unclear. It is possible that both aware and unaware errors occur with some accompanying degree of goal activation, but it is difficult to say if being aware of an antisaccade error equates to a higher or lower level of goal activation compared to an unaware error.

Recent race model accounts of antisaccade performance suggest a strong relationship between speed and accuracy in antisaccade performance (Massen, 2004; Munoz & Everling, 2004). According to these models, at target onset, a race develops between neural activity in the separate prosaccade and antisaccade pathways. A manipulation which increases antisaccade activity (without increasing prosaccade activity), would increase the likelihood of antisaccade activity reaching the threshold for triggering a saccade first. As a result, correct antisaccade latencies and antisaccade errors are reduced. By the same token, a manipulation that increases correct antisaccade latencies should result in an increase in antisaccade error rates providing it does not also increase prosaccade latencies to the same extent. In support of race model predictions, both correct antisaccade latencies and antisaccade errors were increased when participants completed antisaccades with spatial tapping (ST). In contrast, the random number generation (RNG) task manipulation had no effect on correct antisaccade latencies, but still resulted in an increase in error rates, suggesting that the added requirement of spatially tapping keys meant that neural activity in the antisaccade pathway may have been reduced. However, only antisaccade errors were increased (not latencies) when participants completed antisaccades with RNG. It is possible that both correct antisaccade latencies and errors were affected when the ST task was used and not when RNG was used, because the ST task simply placed more demands on participants' ability to simultaneously perform antisaccades. Support for this came from

participant feedback where the majority agreed that the ST task was generally more difficult to perform with antisaccades.

As mentioned above, race model accounts of antisaccade performance would predict an increase in antisaccade error rate, if there had been an increase in correct latencies too. It is therefore difficult to explain why there was an increase in error rate despite no change in correct latencies for the RNG task. One possibility is that the relationship between latencies and errors is not as tight when antisaccades are performed with certain tasks compared to when antisaccades are performed alone. Indeed, an additional analysis revealed a significant correlation between correct antisaccade latencies and antisaccade errors for the antisaccades alone condition but no correlations between these metrics in the 'dual task' conditions. This finding is difficult for current race models of antisaccade performance to interpret and suggests that the relationship between correct antisaccade latencies and errors may be less closely linked in 'dual task' paradigms.

In conclusion, the present results add to previous research suggesting that "aware" and "unaware" antisaccade errors may have different underlying mechanisms. 'Unaware' antisaccade error rate was unaffected by two manipulations designed to alter the "top-down" influences in the task, suggesting that it may reflect more basic properties of saccade generation. An interesting question for future work is whether aware and unaware antisaccade errors differ on additional saccade metrics.

Article 5 - Individual Differences and Antisaccade Performance

Abstract

In the antisaccade task, participants must avoid looking towards a suddenly appearing target stimulus and instead make a saccade to the mirror image location. Numerous studies have found increased antisaccade errors (erroneous prosaccades made towards the target) in patients with a variety of neurological and psychiatric disorders compared to healthy controls. Importantly, a number of studies have also shown that antisaccade error rate can vary tremendously (0-100%) even within healthy participants. Comparatively little research has attempted to establish whether there are individual differences that could account for this variability in error rate in healthy participants. The present studies correlated antisaccade metrics with performance on a range of cognitive tasks and personality measures in an attempt to identify individual differences that might help explain some of this variance. We replicated previous research finding a modest relationship between working memory capacity and antisaccade performance, but found that performance on tasks designed to tap specifically into spatial working memory processes were not associated with antisaccade measures. Performances on tasks measuring speed of processing were also unrelated to antisaccade performance. In support of previous research we found a modest relationship between schizotypy and the antisaccade task, and measures of impulsivity correlated with correct antisaccade latency, but not error rate. The strongest predictor of an individual's antisaccade error rate was in fact their correct prosaccade latency. This finding is important as it supports current competitive race models of antisaccade performance. Overall, the results suggest that the relationship between antisaccade performance and individual differences in cognitive function and personality traits such as schizotypy is generally weak, and that individual differences associated with basic oculomotor processes such as prosaccade latency may be more important mediators of antisaccade error rate.

Introduction

The antisaccade task is now well established as a powerful tool with which to study cognitive function in healthy participants and cognitive deficits in patient populations. The task contrasts controlled behaviour (a volitional saccade made to the opposite side of a target) with the powerful urge to make a prepotent (erroneous) prosaccade towards the target. A large number of studies have found increased antisaccade error rates and increased correct antisaccade latencies in a variety of psychiatric and neurological patient groups (see Hutton & Ettinger, 2006; Leigh & Kennard, 2004 for reviews), and some researchers believe antisaccade performance may serve as a useful endophenotype for schizophrenia (e.g. Calkins et al., 2004; Radant et al., 2007, although see Brownstein et al., 2003).

Whilst average antisaccade error rates in healthy participants (at around 20%) are routinely lower than those observed in psychiatric and neurological patients (and even the relatives of patients with schizophrenia), there are a number of factors which may limit the utility of antisaccade performance as a marker of genetic vulnerability to psychiatric disorders such as schizophrenia. Research has shown that antisaccade performance is not necessarily stable within individuals. For example, Taylor & Hutton (2009) investigated the effects of minor variations in task instructions on antisaccade performance in healthy participants. They found that average antisaccade error rate could differ by as much as 15% between conditions, depending simply on whether participants were instructed to make saccades as quickly as possible or instructed to delay making a response. Other research has found consistent practice effects in antisaccade performance (e.g. Ettinger et al., 2003; Rycroft et al 2007), further suggesting antisaccade error rates may not be particularly stable within participants.

In addition to this within-participant variance, another factor that may limit the utility of antisaccade performance as an endophenotype of schizophrenia or other psychiatric disorders is the very large variance in performance that exists between healthy participants. Although the average error rate for healthy participants is typically around 20%, the standard deviations, when reported in large control samples, are often in the range of 13% - 17% (e.g. Curtis et al., 2001; McDowell et al., 1999). In a large scale study Evdokimidis et al. (2002) measured antisaccade performance in a sample of 2006 healthy male participants and found an average error rate of 23% (*SD* = 17%) and

a range of 0-100%. The enormous range of performance within healthy participants is particularly problematic for researchers interested in using antisaccade performance to measure genetic liability to schizophrenia as well as researchers interested in identifying "at risk" individuals at an early stage in the illness because increased standard deviations mean that larger mean differences in performance are required in order for comparisons across groups to become significant statistically (see for example the meta-analysis of Levy et al., 2008).

Despite the scale of the variability in antisaccade performance in healthy participants, and its relevance to clinical research issues, there has been comparatively little research aimed at identifying individual differences within healthy participants that might explain this variance. Given the extensive literature detailing increased antisaccade errors in patients with schizophrenia, one obvious potentially relevant factor in healthy participants is schizotypal personality traits. According to 'dimension' models of psychopathology (e.g. Claridge, 1997) patients with schizophrenia represent the extreme end of a spectrum of schizotypal personalities. A number of studies have found increased antisaccade error rate in healthy participants who score highly on measures of schizotypal personality traits compared to low scoring individuals (e.g. Ettinger et al., 2005; Gooding, 1999; Holahan & O'Driscoll, 2005; Larrison, Ferrante, Briand & Sereno, 2000; O'Driscoll, Lenzenweger, & Holzman, 1998). O'Driscoll et al. (1998) investigated the relationship between schizotypy and antisaccade performance in a sample of healthy participants. High and low schizotypes were determined by high or low scores respectively on the Perceptual Aberration Scale (Chapman et al., 1978). This scale focuses on the positive symptoms of schizotypy, such as perceptual distortions. Only step antisaccades were used, where the offset of the fixation point coincided with the onset of the peripheral target. The authors found that low Schizotypes had a higher percentage of correct antisaccades compared to high Schizotypes. However, there was no difference between the groups in terms of correct antisaccade latencies. In another study, Gooding (1999) used both the Perceptual Aberration Scale and the Social Anhedonia Scale, a measure of more "negative" schizotypal personality traits (Chapman et al 1994) to investigate the effects of schizotypy on prosaccade and antisaccade performance. Task performance was compared between healthy controls and participants who scored highly on each scale. Again, only step antisaccades were used. The authors found no difference in prosaccade performance between groups, nor any differences in correct antisaccade latencies between groups. However, participants in

the schizotypy groups made significantly more antisaccade errors than healthy controls, although the difference between groups was only 4%.

Larrison et al. (2000) used the Schizotypal Personality Questionnaire (SPQ) (Raine, 1991) to investigate the relationship between schizotypy and eye movements. The SPQ includes subscales concerned with both the positive and negative symptoms of schizotypy. Using step and gap versions of the antisaccade task, the authors found that high scoring Schizotypes had increased antisaccade errors compared to low scoring Schizotypes on the gap but not the step version. They found no relationship between schizotypy and correct antisaccade latencies. In a more recent study, Ettinger et al. (2005) found that high positive schizotypy scores (as measured by the Rust Inventory of Schizotypal Cognitions, which taps positive schizotypy symptoms; Rust, 1989) correlated significantly with antisaccade errors on step trials, in a large sample of healthy participants. However, in support of previous work (Gooding 1999; O'Driscoll et al., 1998) there was no relationship between schizotypy and correct antisaccade latencies. Finally, Holahan & O'Driscoll (2005) explored the relationship between schizotypy and antisaccade performance using the Perceptual Aberration Scale and the Physical Anhedonia Scale (Chapman et al., 1976). The Physical Anhedonia Scale is designed to tap negative symptoms of schizotypy. Step, overlap and gap versions of the antisaccade task were used. In support of past work (Gooding, 1999; O'Driscoll et al., 1998), the authors found increased antisaccade errors in positive symptom Schizotypes (as measured by the Perceptual Aberration Scale) and increased errors in negative symptom Schizotypes (as measured by the Physical Anhedonia scale) compared to healthy controls only on the step version of the antisaccade task, not overlap or gap. There were no differences in correct latencies between controls and schizotypy groups for all versions of the antisaccade task.

To date, studies investigating the relationship between schizotypal personality traits and antisaccade performance have revealed two key results: Firstly, all studies have reported that increased schizotypy is associated with increased antisaccade errors in healthy participants. Secondly, in these studies correct antisaccade latencies are unaffected by schizotypy. It is interesting to note however, that there are some inconsistencies between studies. For example, Larrison et al. (2000) found an effect of schizotypy on error rate using a gap version of the antisaccade task whereas Holahan & O'Driscoll (2005) only found a difference using a step version. There is also very little

consistency across studies in terms of the scales used to measure schizotypal personality traits.

Another individual difference that might account for some of the variance in antisaccade performance observed across healthy participants is working memory capacity. According to 'goal activation' accounts of antisaccade performance (e.g. Eenshuistra, Ridderinkhof, & Van der Molen, 2004; Hutton, 2008; Nieuwenhuis, Broerse, Nielen, & de Jong, 2004; Roberts, Hager, & Heron, 1994) antisaccade errors are more likely to occur if the goal (to make a correct response) is not sufficiently activated within working memory. Several studies have explored the relationship between working memory and antisaccade performance in healthy participants. In an early study Roberts et al. (1994) gave participants a block of antisaccades to complete followed by a second block of antisaccades with an additional task to complete. In this second block, participants had to perform mental arithmetic (adding current sum to a new number) whilst simultaneously performing antisaccades. Participants had increased antisaccade errors and correct antisaccade latencies when completing both mental arithmetic and antisaccades compared to performing antisaccades alone.

In a more recent paper, Mitchell, Macrae, & Gilchrist, (2002) also used a dual task approach to explore the relationship between working memory and antisaccade performance. In their first experiment, they gave healthy participants' blocks of antisaccades only and blocks of antisaccades with an additional task (the n-back task, Jonides et al., 1997). In the n-back task, participants were presented with letters of the alphabet via computer speakers. Participants had to decide if the currently presented letter matched the target letter specified by the experimenter. For example, if 'P' was presented and it was the target letter, then the participant had to press the computer key that corresponded to a 'yes' response. In a variation of the task, participants had to decide if each presented letter matched the preceding letter or not (1-back). Similarly, they also asked participants to perform another variation of the n-back task (2-back) where participants had to decide if the currently presented letter matched the letter from two previous letters in the series. The results showed that compared to the antisaccade only condition, there was no increase in antisaccade errors when the 0 n-back or 1 n-back task was performed with antisaccades. However, errors were increased when participants performed the 2 n back task with antisaccades. In contrast with Roberts et al. (1994), there was no increase in correct latencies when both tasks were performed. This absence of an effect on latencies may have occurred due to a lack of statistical

power because Mitchell et al used very few participants (N = 16). Taken together these results provide some support for a relationship between working memory and antisaccade performance.

Given the role of working memory in antisaccade performance suggested by goal activation accounts (and supported by the dual-task studies outlined above), and the large body of research detailing individual differences in working memory capacity (e.g. Daneman & Carpenter, 1980; Kane & Engle, 2003) it is unsurprising that researchers have explored individual differences in working memory capacity as a possible predictor of antisaccade performance. Kane, Bleckley, Conway, & Engle (2001) for example compared prosaccade and antisaccade performance in high working memory span and low working memory span participants. Working memory span was measured using the OSPAN task, in which participants are asked to read aloud a simple maths problem (such as 3 + 5 = 7?) and decide if the answer is true or false. Immediately afterwards, participants read aloud an unrelated word that is to be recalled at a later time. This continues until three question marks are presented, at which point participants are asked to recall the remembered words in that set. Each set consisted of 2-6 maths problems/words. 15 sets of problems were presented in total. OSPAN scores were the sum of the recalled words for all sets recalled completely and in correct order. High span participants were those who were in the top quarter of the distribution of OSPAN scores and low span participants were those from the bottom quarter. In both the prosaccade and antisaccade tasks, participants were required to make either a prosaccade or antisaccade as quickly and as accurately as possible, and then identify a pattern masked letter (B, P or R) which appeared at the goal location. When the target letter was identified, participants had to respond by pressing the corresponding key on the keyboard. The study found equivalent performance between high and low span individuals when participants had to make a prosaccade and identify a target letter. However, performance between high and low-span individuals differed when an antisaccade was required to the identified target letter. High-span individuals made fewer errors and made faster correct antisaccades than low-span individuals on this task. The authors concluded that low-span individuals are more susceptible to goal neglect, suggesting that task-relevant information is not continuously activated and maintained, causing a decline in antisaccade performance. The results of Kane et al's study suggest that individual differences in antisaccade performance in healthy participants can be explained to some extent by individual differences in OSPAN scores.

More recently, Unsworth, Schrock, & Engle (2004) also found differences in antisaccade performance between high and low-span individuals. Again, using the Operation Span task (OSPAN) to determine high and low-span participants, they observed slower correct antisaccade latencies and more errors in low-span compared to high span participants. In addition, the authors found a negative correlation between OSPAN scores and antisaccade error rate in healthy participants, contrary to past work that has failed to find a relationship between antisaccade errors and other working memory tasks in healthy participants (Hutton et al., 2004).

Although the OSPAN task has been established as a predictor of antisaccade error rate (Unsworth et al., 2004) in the present paper, as well as using the OSPAN task, we will consider additional measures of working memory as potential correlates of antisaccade performance. Some studies have found correlations between measures of working memory span and antisaccade performance in clinical samples. Nieman et al. (2000) used a spatial working memory test to explore the relationship between working memory and antisaccade performance in Schizophrenic patients. On the spatial working memory test, participants were shown a spot on the computer screen. After the spot disappeared, random words were shown on screen and had to be read aloud. After several minutes, the participant had to point to the location on the screen where the spot had been shown. Scores were calculated as the average distance between the various locations of the displayed spot and each location that was pointed to after its disappearance. The authors found a positive correlation between antisaccade error rate and scores on the spatial working memory test, that is, as antisaccade errors increased, so did errors on the spatial working memory test.

A relationship between spatial working memory and antisaccade performance in patients with schizophrenia was also found in a more recent study. Hutton et al. (2004) used the spatial span task (Owen et al., 1990), to investigate this relationship, where sequences of squares are presented on screen and participants have to remember the order of sequences. The authors reported a positive correlation between antisaccade performance and performance on the spatial span task in a sample of Schizophrenic patients. That is as antisaccade errors increased, so did spatial span errors. The authors also correlated performance on the spatial working memory task (Owen et al., 1990) with antisaccade performance. In the spatial working memory task, participants must find tokens by opening sets of boxes varying in set size (3-8 boxes). An error is made if a box that contained a token is re-opened. From this task, the authors also recorded a

measure of strategy, where that a common strategy employed is to follow a predetermined search sequence beginning with the same box. A higher score for this measure indicated a poorer use of strategy. Spatial working memory errors and strategy scores both correlated positively with antisaccade errors and these correlations were stronger compared to the correlation between spatial span and antisaccade errors. In addition, a regression analysis revealed that spatial working memory errors were the best predictor of antisaccade errors. The authors concluded that spatial working memory errors were the best predictor of antisaccade errors because this is the measure that reflects most directly the integrity of working memory processes. Taken together, these results suggest that tasks that measure spatial working memory are related to antisaccade error rate in Schizophrenic patients. Moreover, these results suggest that processes involved in spatial working memory may be particularly relevant to antisaccade performance, at least in schizophrenia. Therefore, it is possible that this relationship between spatial working memory and antisaccade performance could be extended in healthy participants.

Although research has focused on the effects of schizotypy on antisaccade performance, individual differences in other personality traits may also be relevant. Impulsivity is a personality trait that according to Claridge (1995) is a specific sub-component of schizotypy. It has also been argued to play a critical role in a number of psychiatric disorders such as attention deficit hyperactivity disorder (ADHD), in which antisaccade errors are known to be increased (e.g. Mostofsky, Lasker, Cutting, Denckla, & Zee 2001). Keilp, Sackheim, & Mann (2005) explored the relationship between impulsivity and a range of neuropsychological tests using healthy participants. The authors used the Barratt Impulsiveness Scale (BIS) to measure impulsivity. They found that impulsivity scores correlated with measures of fluency and executive functioning. In general, higher impulsivity scores were associated with slower or poorer performance across a range of tasks. Other studies have also reported deficits in cognitive task performance in impulsive populations. For example, Potter & Newhouse (2004) found decreased performance on the Stroop task (Stroop, 1935) in adolescents with ADHD, a disorder associated with high impulsivity characteristics. Performance on both the Stroop task and antisaccade task requires a degree of response control therefore it is possible that impulsivity could also relate to antisaccade performance. A further aim of the current paper was to extend the range of personality measures that have been found to influence antisaccade performance in healthy participants.

As mentioned earlier, one view is that correct antisaccade performance depends critically on keeping the intention to make the antisaccade sufficiently active within working memory (Nieuwenhuis et al., 2004). However, other requirements may play a key role in the outcome of the antisaccade. The speed with which correct antisaccades can be initiated, for example could impact on the accuracy of the antisaccade. Competitive race models of antisaccade performance (Massen, 2004; Munoz & Everling, 2004; Reuter & Kathmann, 2004) highlight the importance of 'speed of processing', as they suggest at target onset, a race ensues between activity in the separate prosaccade and antisaccade pathways, with the winner reaching the threshold for triggering a saccade first. Support for race accounts of antisaccade performance comes from observed relationships between prosaccade latency and antisaccade error rate (e.g. Roberts et al., 1994). The authors reported a negative correlation between these measures, that is, increased prosaccade latencies correlated with reduced antisaccade errors (those who were fastest with prosaccades were least accurate with antisaccades) These findings imply the importance of 'speed' to antisaccade performance.

Given the relationship between prosaccade latency and antisaccade error rate within individuals, it is possible that antisaccade error rate could be associated with other tasks that measure 'speed of processing'. Speed of processing tasks can be divided into two broad categories, 1) 'motor speed' and 2) 'internal processing speed'. Tasks that are concerned with performing a motor operation as quickly as possible (e.g. finger tapping task, Reitan & Wolfson, 1985) fall into the 'motor speed' category, whereas tasks concerned with performing internal cognitive operations as quickly as possible (e.g. the letter comparison task (Salthouse & Babcock, 1991) fall into the 'internal processing speed' category. An additional aim of this paper was to see if either type of speed of processing task would correlate with antisaccade performance.

In this paper we describe 3 analyses, each of which aimed to identify potential correlates of antisaccade performance, in an attempt to identify individual differences that might explain the very high variability in antisaccade error rate typically observed even within healthy participants. In the first study, we sought to replicate and extend previous demonstrations of associations between antisaccade performance and measures of working memory. In addition to the widely used OSPAN measure, we also measured spatial working memory using two additional measures of working memory and response inhibition using the go-no go task. In the second study, prompted by

competitive race accounts of the antisaccade task, we explored the relationship between antisaccade performance and a range of measures of "speed of processing". In the final study, we sought to replicate and extend previous research demonstrating associations between various personality measures and antisaccade performance.

The following reported analyses were conducted on data taken alongside the antisaccade data reported earlier in other papers in this thesis. Individual differences were always of interest and for clarity, all these aspects of the data are considered together here in a single paper, rather than as separate analyses within individual papers.

Analysis 1(working memory)

As mentioned previously, previous research exploring the relationship between working memory and antisaccade performance has tended to use the operation span (OSPAN) task (e.g. Kane et al., 2001; Unsworth et al., 2004), and whilst significant associations have been found, they have not been large. In addition to using the OSPAN task, we wanted to see if other measures of working memory capacity would be better predictors of antisaccade performance in healthy participants. On a typical antisaccade trial, the co-ordinates of the exact goal location must be calculated in order for an accurate saccade away from the impending target to be successful, thus the task has a spatial element. In addition, research in patients with schizophrenia has observed correlations between antisaccade performance and measures of spatial working memory (e.g. Hutton et al., 2004). Therefore to attempt to find better predictors of antisaccade performance we included the matrix span task (MSPAN) and the symmetry span task (SSPAN), as these tasks tap spatial working memory processes which may be more relevant to antisaccade performance than the processes tapped by the OSPAN task.

Inhibition is an important aspect of working memory. According to goal activation accounts of antisaccade performance inhibition of an erroneous prosaccade occurs as a by-product of activating and maintaining the goal (making a correct antisaccade to the opposite location of the target) (e.g. Cutsuridis, Smyrnis, Evdokimidis, & Perantonis, 2007; Nieuwenhuis et al., 2004; Roberts et al., 2004). An additional aim of the first analysis was to see if performance on a go-no go task would correlate with antisaccade performance. We aimed to replicate the correlation between go-no go performance and antisaccade performance found by Spinella (2002), using more trials

and a greater sample size. We expected to find a relationship between the tasks as they are both concerned with response inhibition and share neural anatomical substrates.

Method

Participants

For OSPAN analysis, the 58 participants described in article 1 (exp. 1) were used here. For MSPAN and SPSAN analyses, the 35 participants described in article 3 (exp. 1) were used. For the go-no go analysis, 26 participants were taken from an experiment that was not included in the thesis. These participants were all students from the University of Sussex of whom 18 were female and 8 were male. Ages ranged from 19-32 (M = 23.09, SD = 4.71). All participants provided written consent and were naïve to the purpose of the study.

Apparatus

Eye movements were recorded with an Eyelink II eye tracker (SR-Research Ltd. Ontario, Canada), with a spatial resolution of about 0.25 degrees and a temporal resolution of 2ms. Participants used a modified computer chair that prevented any rotational movement, seated 70cm from the computer screen. A 21 inch CRT monitor was used to display the stimuli, operating with a refresh rate of 100Hz and a resolution of 1,280 x 1,024 pixels. A brief 9 point calibration was done, prior to the experiment and repeated if necessary between blocks.

Antisaccade tasks stimuli/measures/procedures

Descriptions of stimuli/measures can be found in article 1 (exp. 1) for OSPAN and article 3 (exp. 1) for MSPAN and SSPAN. Antisaccade task stimuli/measures used for go no-go analysis was identical to that used in article 3 (exp. 1) except there were only 2 goal locations. In article 1 (exp. 1) participants' performed a mixed prosaccade and antisaccade task in which 66% of trials also contained a cue. To ensure that only the most straightforward correlations were performed, OSPAN performance was correlated with antisaccade error rate and correct antisaccade latencies for the uncued trials (N trials = 64). In article 3 (exp. 1), participants performed antisaccades under different task instructions. Again, so that only straightforward correlations were performed,

MSPAN and SSPAN performance was correlated with antisaccade error rate and correct antisaccade latencies for standard instructions trials (N trials = 64). Finally, go-no go performance was correlated with standard errors and correct latencies (N trials = 80). Participants who performed the go-no go task also performed 4 blocks of antisaccade trials under differing instructions, standard, accuracy, speed, delay (see paper 3 for descriptions) where only 2 possible goal locations were used (+/- 10 degs from the centre point)

Operation span task (OSPAN)

The Operation span task (Turner & Engle, 1989) requires participants to solve a series of operation-word problems, whilst trying to remember a set of unrelated words. For each problem, located on the centre of a computer screen, participants are required to read aloud and give a Yes/No response to a simple maths problem and then read an adjacent unrelated word out loud. There are 12 blocks of items - three blocks each of 2, 3, 4 & 5 items. After each block, the participant is required to recall as many of the words as possible. For example, a two-item block could be as follows:

IS (7 x 3) - 2 = 4? : CAT,

IS (6 + 2) + 1 = 7? : TREE,

???

Figure 5.1. An example of a trial on the OSPAN task

The first operation-word problem is shown and remains on screen until both the maths part has been solved (the participant answers yes or no) and the unrelated word has been read aloud. The first item is then replaced by the second item and the process is repeated. Finally, the second problem disappears and three question marks appear, which indicate that it is now time for the participant to try to recall as many of the words from that block, in the same order in which they were encountered. Participants are required to write down the recorded words. Blocks consisted of 2-5 operation-word problems and scoring was achieved by totalling the number of correctly recalled words over all blocks.

Matrix span task (MSPAN)

In the matrix span task (Kane et al., 2004) participants were shown several 4 x 4 matrices, containing 16 squares. The task was to remember where the one red square was in the matrix and place a cross in the corresponding square on the answer sheet. There were 18 sets that may contain between 2-7 matrices. Participants had as long as they needed to answer, but had to recall each red square in the order the matrices were presented, for example:

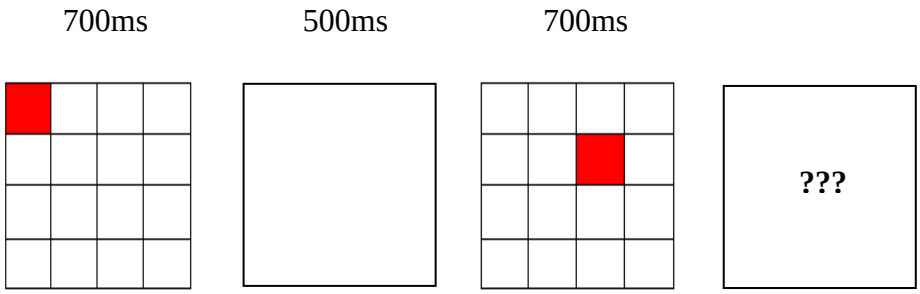

Figure 5.2. An example of a trial on the MSPAN task

On presentation of the question marks, participants recorded where they thought the red squares appeared, in order. The number of correct items in a set, were converted into a proportion score, e.g. Set 1: 2/5 = .40, Set 2: 4/7 = .57. Then the mean proportion correct score was calculated over all sets to make a matrix span score.

Symmetry span task

The symmetry span task (Kane et al., 2004) required participants firstly to decide if a pattern presented in an 8 x 8 matrix was symmetrical, then secondly, to remember the location of one red square from a 4 x 4 matrix. Participants answered aloud (yes or no), immediately after the pattern square had been presented, if they thought it was symmetrical on the vertical axis. The experimenter then pressed a key that produced a blank screen for 500ms, which was followed by a to-be remembered red square matrix. The process was repeated until question marks appeared, then participants tried to recall as many red squares in order from the matrices they had just seen, by placing a cross in the corresponding box on the answer sheet.

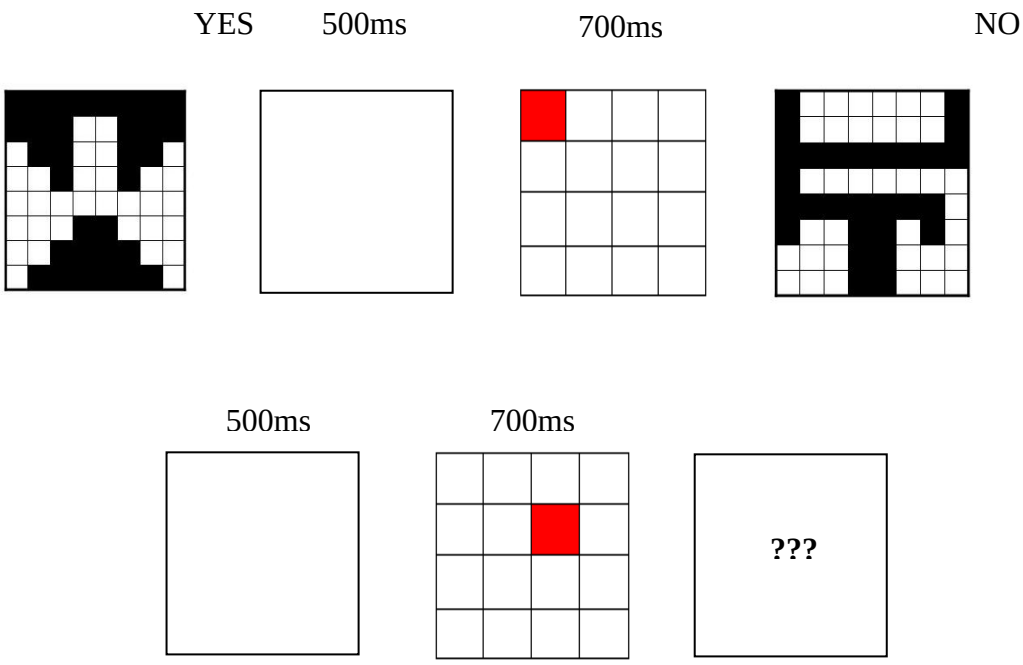

Figure 5.3. An example of a trial on the SSPAN task

A proportion correct score was calculated, the same way it was calculated in the Matrix span task.

Go-no go task

In an adapted version of the go-no go task (Nieuwenhuis et al., 2003), participants were required to press the "G" key upon seeing the stimulus corresponding to "go" (the letter M) and inhibit pressing the keyboard upon seeing the "no go" stimulus (the letter W). Participants sat 70cm from the screen and stimuli were presented in red, on a white background. Stimuli were shown for 100msec with a varying inter-stimulus interval ranging from 1.1 to 1.9 seconds. The task comprised 144 go and 36 no go trials, with no practise trials given. Errors of omission occurred when participants failed to press the G key when presented with the go stimulus and errors of commission occurred when participants erroneously pressed the key when presented with the no-go stimulus (failure to inhibit).

Results

All correlations are 2 tailed unless stated otherwise.

Operation span task

On average, participants' OSPAN scores were 24.33 (*SD* = 6.74), with scores ranging from 11 - 42. Average antisaccade errors were 25%, (*SD* = .20), ranging from 0 - 91%. Average correct latencies were 278msec (*SD* = 42.31, range = 170msec – 391msec).

OSPAN scores correlated with the percentage of uncued antisaccade errors (*r* = -.24, *p* = .03, 1 tailed). A higher OSPAN score was associated with fewer antisaccade errors.

There was no correlation between OSPAN scores and uncued correct latencies *r* = - .00, *p* = .99.

Matrix span task

On average, participants' MSPAN scores were 60.77 (*SD* = 11.46, range = 28.9 -81.1). Average antisaccade errors were 33% (*SD* = .21, range = 03% - 75%). Average correct latencies were 245msec (*SD* = 45.09, range = 176msec - 344msec).

MSPAN scores failed to correlate with percentage of antisaccade errors and correct latencies under standard instructions (*rs* > -.04, *ps* > .20).

Symmetry span task

On average, participants' SSPAN scores were 51.78 (*SD* = 16.37, range = 17.9 - 84.6).

SSPAN scores failed to correlate with percentage of antisaccade errors and correct latencies under standard instructions (*rs* > -.02, *ps* > .78).

Go- no go task

On average, participants made significantly less errors of omission (*M*= 1.07, *SD*= 1.18) than commission (*M*= 6.88, *SD*= 5.49), on the go-no go task. Participants average antisaccade error rate was 36%, (*SD* = .20, range = 06% - 82%). Average correct latencies were 201.07msec (*SD* = 49.84, range = 112.77msec – 299.90msec).

Errors of omission correlated with percentage of antisaccade errors under standard instructions (r = .32, p = .05, 1 tailed), that is, those who did not 'go' when they were supposed to were those who were more likely to make an antisaccade error. Errors of omission failed to correlate with correct latencies under standard instructions (r = .16, p = .45).

The correlation between errors of commission and percentage of antisaccade errors under standard instructions approached significance (r = .31, p = .06, 1 tailed). Errors of commission failed to correlate with correct latencies under standard instructions (r = -.15, p = .44). Errors of commission accounted for 10% of the variance in antisaccade errors.

Discussion

In this first set of analyses we explored the relationship between performance on the antisaccade task and performance on tasks that measure working memory capacity and response inhibition. We replicated previous research showing a relationship between working memory capacity as measured by the OSPAN task and antisaccade error rate. In addition, we demonstrated that antisaccade error rate correlates with errors of omission on a go no-go task. Antisaccade performance was unrelated to spatial working memory capacity, as measured by two different tasks (symmetry and matrix span).

The finding that participants OSPAN scores correlated with antisaccade errors replicates previous research (Unsworth et al., 2004) and supports the hypothesis that correct antisaccade performance requires efficient working memory processes. However, the correlation between OSPAN scores and antisaccade errors was modest. In Unsworth et al's study a large number of participants were screened with the OSPAN task and antisaccade performance was compared only in those participants who scored in the upper quartile and lower quartile of the OSPAN distribution. The OSPAN score of our high group was very close to that of the high group in Unsworth et al. (27.3 vs. 27.9) but our low span group had significantly higher spans (20 vs. 6.1). Thus one possibility is that the relatively modest correlation we observed reflects the fact that the range of working memory capacities, as measured by OSPAN in our sample was relatively restricted.

Although the sample size was relatively small (*N* = 26) participants performance on the go-no go task correlated with antisaccade error rate. Spinella (2002) also found a correlation between antisaccade error rate and performance on a go-no go task, whereby those who made more errors of commission also made more antisaccade errors. In Spinella's go-no go task, participants had to imitate a set of taps performed by the experimenter. If the experimenter tapped the table once, then the participant had to copy this (go) but if the experimenter tapped twice, then the participant had to refrain from imitating this (no-go). Errors of commission on this task occurred if a participant imitated the experimenter if they had tapped twice, or if a response was delayed by more than 1 second.

The finding that spatial working memory (as measured by the MSPAN and SSPAN) failed to correlate with antisaccade performance was unexpected. Previous research, particularly in patients with schizophrenia, suggests that spatial working memory processes may be particularly important for antisaccade performance. Both the MSPAN task and the SSPAN task require a degree of spatial processing as a participant must recall the location of a previously shown red square. Similarly, spatial processing is required when generating an antisaccade, as a participant must calculate the co-ordinates of the goal location of the antisaccade. The range of scores on both the MSPAN and SSPAN tasks varied considerably (MSPAN = 28.9 – 81.1, SSPAN = 17.49 – 18.46), but the sample size for these correlations was relatively small (*N* = 35), which may have limited any chances of observing a significant correlation.

Analysis 2 (speed of processing)

Competitive race models of antisaccade performance suggest that speed of processing may be an important determinant of antisaccade performance. Specifically, such models predict that antisaccade error rates will be highest in those participants who take longest to program a correct antisaccade. In this second set of analyses, we correlated antisaccade performance with performance on a range of tasks indexing processing speed. The first speed of processing task was the letter comparison task (Salthouse & Babcock, 1991). The letter comparison task is well established and widely used in the literature as a general measure of processing speed (e.g. Salthouse, 1993). The second task was the Digit symbol substitution task (DSST) (Wechsler Adult

Intelligence Scale-Revised, Wechsler, 1981). The digit symbol substitution task (DSST) has also been widely used as a general measure of processing speed in a range of settings (e.g. Glosser, Butters & Kaplan 1977; Morgan & Wheelcock, 1995). The third task was the finger tapping task (Reitan 1969). The finger tapping task (FTT) is generally considered to be a measure of "psychomotor speed" (e.g. Archbold, Borghesani, Mahurin, Kapur, & Landis 2009). Previous research has found a relationship between correct prosaccade latencies and antisaccade errors, highlighting the importance of speed in antisaccade performance. The correlation between prosaccade latency and antisaccade error rate found by Roberts et al. (1994) suggests that those who make fast prosaccades are those who make fewest antisaccade errors. In the following analyses we attempted to replicate the correlation between correct prosaccade latency and antisaccade errors and also predicted that other measures of general cognitive speed, as indexed by the letter comparison task, digit symbol substitution task and the finger tapping task, would also correlate with antisaccade error rate.

Method

Participants

For the main analysis, the 26 participants described in article 3 (exp. 2) were used. We also performed a supplementary analysis on the relationship between correct prosaccade latency and antisaccade error rate. This analysis was performed on the 58 participants in article 1 (exp. 1). Further details are provided below.

Antisaccade procedures

As with the earlier analyses, letter comparison scores, digit symbol substitution scores and finger tapping scores were correlated with antisaccade errors and correct antisaccade latencies for standard instructions trials from article 3 (exp. 2). For the supplementary analysis, uncued prosaccade latencies were correlated with uncued antisaccade errors (article 1, exp. 1) and also between standard prosaccade latencies and standard antisaccade errors (article 3, exp. 2).

Letter comparison task

The letter comparison task (Salthouse & Babcock, 1991) consisted of two pages containing pairs of random consonants, of either three, six or nine letters in size. Participants were given 30 seconds for each page to indicate if they thought the pair was the same or different. The letter 'S' was written down if they thought the pair matched and the letter 'D', if they did not think it matched. One half of the pairs on each page were the same and one half were different. One point was given for every correct answer and one deducted for an incorrect response.

GDX **S** GDX

KGDXNMYRW **D** KGDXFMYRW

PKYXMF **S** PKYXMF

Figure 5.4. An example of a trial from the letter comparison task

Digit symbol substitution task (DSST)

The digit symbol substitution task (from the Wechsler Adult Intelligence Scale-Revised Wechsler, 1981), comprised one page of 118 randomized numbers between 1 and 9. Using the key provided at the top of the page, for each number, participants were required to draw its corresponding symbol underneath the number and repeat this process for all following numbers. Participants were given 90 seconds to attempt as many as possible and one point was given per correct answer.

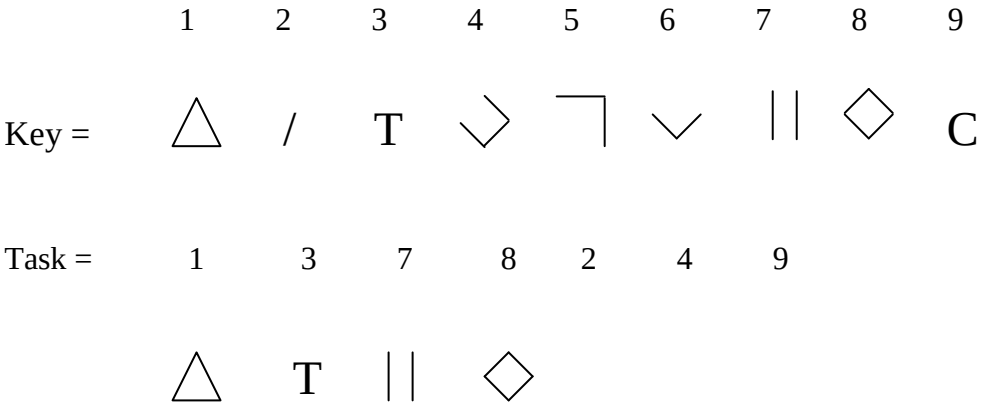

Figure 5.5. An example of a trial from the DSST

Finger tapping task (FTT)

In the finger tapping task, the instructions were derived from Reitan & Wolfson (1985). Participants were required to see how many times they could press the zero key on a standard PC. There were 5 blocks, each lasting 10 seconds and the sub total from each block was added to make the total score.

Results

Multiple regression analysis

In the previous analysis, two different samples of participants were used, so separate correlations were performed between antisaccade measures and the various working memory measures. In this analysis the same participants were used for all speed of processing tasks, therefore a multiple regression was conducted in order to see if scores on the letter comparison task, DSST and the FTT were significant predictors of antisaccade error rate. All speed of processing tasks were entered in one block using forced entry method. The outcome variable was standard antisaccade errors.

The final model was not a significant fit of standard antisaccade errors ($F(3, 21) = .78$, $p = .52$, $r = .19$) and explained 10% of the variance in antisaccade error rate. There were no significant predictors of antisaccade error rate ($ps > .15$). The strongest predictor of antisaccade errors was the DSST ($\beta = -.36$).

Correct prosaccade latencies

In two of the experiments conducted during this thesis the same participants performed both prosaccade and antisaccades. Correlations were conducted between correct prosaccade latencies and antisaccade errors in these samples. The uncued prosaccade and antisaccade data was from article 1 (exp. 1, $N = 58$) and the standard prosaccade and antisaccade data was from article 3 (exp. 2, $N = 26$).

The correlation between correct prosaccade latencies and uncued antisaccade errors in the sample from article 1 (exp. 1) was significant, ($r = -.60$, $p < .001$). In other words, those who were fastest to make a prosaccade were more likely to make an antisaccade error (see figure 5.6). Prosaccade latency accounted for 36% of the variance in uncued antisaccade errors.

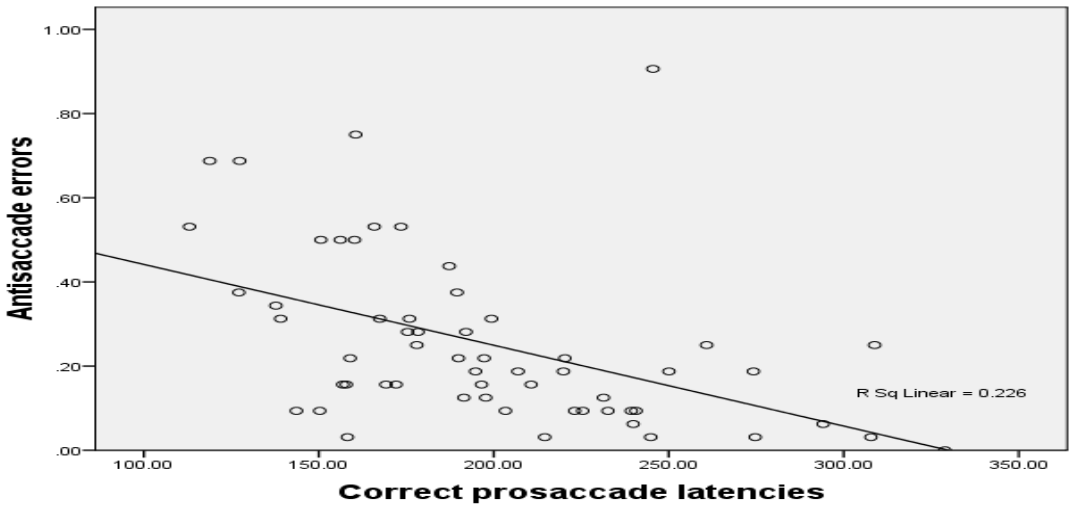

Figure 5.6. Correlation between antisaccade error rate and prosaccade latencies (article 1, exp. 1)

Interestingly, this correlation was not replicated in the other smaller sample from article 3 (exp. 2), (r = -.20, p = .32).

Discussion

In the second analysis, we investigated the relationship between antisaccade performance and performance on three speed of processing tasks. Contrary to our predictions, participants' scores on the letter comparison task, digit symbol substitution task and finger tapping task failed to correlate with antisaccade error rate. The only measure of "speed of processing" which correlated with antisaccade performance was correct prosaccade latency, and even this was only significant in one sample.

There are other studies that have observed modest correlations between prosaccade latency and antisaccade error rate (e.g. Roberts et al., 1994). Indeed, we only observed this relationship between prosaccade latency and antisaccade errors in one of our data sets and not the other. Importantly, the two data sets we used had different designs (prosaccades and antisaccades were performed in mixed blocks in the significant sample and separate blocks in the non significant sample. It is possible that the strong correlation we observed in the first data set (N = 58) reflects the fact that in the mixed design, where the status of each trial was not known until the cue changed to red (antisaccade) or green (prosaccade), activity in the prosaccade pathway remained

comparatively high throughout the task, thus increasing its relevance to antisaccade performance. Another factor is that the sample size in the significant analysis is larger (58 vs. 26).

Analysis 3 (Personality)

In the third set of analyses we examined the relationship between measures of personality and antisaccade performance. Previous research suggests that questionnaire identified schizotypal participants show poorer antisaccade performance compared to healthy controls (e.g. Ettinger et al., 2005; Gooding, 1999). To measure schizotypy, we used the Oxford Liverpool Inventory of Feelings and Experiences (OLIFE) (Mason, Claridge & Jackson, 1995). The OLIFE is a widely used questionnaire (e.g. Avons, Nunn, Chan, & Armstrong, 2003) and has shown high internal consistencies for all four sub scales (α's > .77, Mason et al., 1995). Many studies that have looked at the relationship between schizotypy and antisaccade performance have used the Perceptual Aberration-Magical Ideation Scale (e.g. Gooding, 1999; Holahan & O'Driscoll, 2005; O'Driscoll et al., 1998). However, this scale only takes into account positive symptoms of schizotypy. An advantage of using the OLIFE is that it contains sub scales that are relevant to both the positive (unusual experiences) and negative (introvertive anhedonia) symptoms of schizotypy. Therefore in the present analysis, we are able to look at questionnaire identified Schizotypes who show positive and or negative symptoms of schizotypy and how this relates to antisaccade performance. In addition, the OLIFE is a more versatile questionnaire, as it contains two additional sub scales designed to find symptoms of cognitive disorganisation and impulsive non-conformity. The cognitive disorganisation scale is also concerned with positive symptoms of schizotypy, whereas the impulsive non-conformity scale is concerned with asocial aspects of schizotypy and psychoticism. This dimension of the OLIFE is useful for finding participants who may be susceptible to developing psychosis. In short, the OLIFE is a robust instrument for measuring schizotypy as it is based on the most extensive investigation of schizotypal traits using in excess of 1000 participants (Mason & Claridge, 2006). To our knowledge, this will be the first study to use the OLIFE as a potential correlate of antisaccade performance.

In addition, we explored the potential relationship between impulsivity and antisaccade performance. The relationship found between schizotypy and the antisaccade task in previous studies may extend to impulsivity as impulsivity is one characteristic of schizotypy. Again, we were interested to know if impulsivity scores could account for any variability in antisaccade error rate. To measure impulsivity, we used the Barratt Impulsiveness Scale (BIS) (Patton, Stanford, & Barratt, 1995). The BIS has good reliability, with Cronbachs alpha's ranging from α .79 – .83 (see Patton et al., 1995), making it more than suitable for use as a tool for measuring impulsivity in non-clinical populations. The questionnaire has been used in antisaccade studies before. For example, Spinella (2004) found a correlation between BIS scores and antisaccade error rate in a sample of healthy participants.

Based on past work, it was expected that there would be a correlation between schizotypy (as measured by OLIFE) and antisaccade performance, with higher schizotypy scores associated with higher error rates. Similarly, we expected a relationship between impulsivity (as measured by BIS) and antisaccade performance, participants with high BIS scores will make more antisaccade errors and take longer to make a correct antisaccade than participants with low BIS scores.

Method

Participants

For the schizotypy analysis, the 58 participants described in article 1 (exp. 1) were used here. For the Impulsivity analysis, the 26 participants described in article 3 (exp. 2) were used.

Antisaccade procedures

Antisaccade procedures are taken from the above corresponding papers. Importantly, correlations were contrasted between OLIFE scores and uncued antisaccade errors and correct latencies. BIS scores were correlated with standard antisaccade errors and correct latencies.

Oxford Liverpool Inventory of Feelings and Experiences (OLIFE)

Schizotypal personality traits were measured with the Oxford-Liverpool Inventory of Feelings and Experiences (Mason, Claridge, & Jackson, 1995). The questionnaire consists of 104 items, divided into four subscales: (i) Unusual experiences (positive) (ii) Cognitive disorganization, (disorganized) (iii) introvertive anhedonia (negative) (iv) impulsive non-conformity (psychoticism). Each item consists of a statement such as "Have you felt that you have special, almost magical powers?" and respondents respond by circling either yes or no. A 1 is given for yes and 0 for no. Some items are reverse scored. The unusual experiences subscale measures "positive" aspects of schizotypal personality such as odd beliefs /magical ideation. Cognitive disorganization contains items that concern cognitive difficulties such as problems with concentrating and decision making as well as emotional sensitivity and social anxiety. The items in the introvertive anhedonia subscale measure "negative" aspects of schizotypal personality such as lack of enjoyment in social contact. The impulsive non-conformity subscale contains items concerned with asocial and impulsive behaviour.

Barratt Impuliveness Scale (BIS)

Participants completed the 30 item BIS, by responding either as “rarely/never, occasionally, often, or almost always/always”. Questions were related to things such as extraversion and sensation seeking, for example, “I act on impulse, or I often have extraneous thoughts when thinking”. Scoring was calculated by applying a 1 for rarely/never, through to 4 for almost always/always.

Results

Schizotypy

Total OLIFE scores did not correlate with uncued percentage of antisaccade errors (r = .06, p = .68), or with uncued correct antisaccade latencies, (r = -.06, p = .64). Scores for the unusual experiences, cognitive disorganization, and introvertive anhedonia subscales of the OLIFE did not correlate with any of these antisaccade metrics (rs < .28, ps > .08). The correlation between scores on the impulsive non-conformity scale and antisaccade error rate approached significance (r = .20, p = .06, 1 tailed).

Impulsivity

Scores on the Barratt Impulsivity Scale (BIS) correlated significantly with standard correct antisaccade latencies (r = .46, p = .01), suggesting that a high score corresponds with having a slower speed of response to make a correct antisaccade. However, the relationship between BIS scores and standard antisaccade errors was not significant (r = -.04, p = .85) and there was no correlation between BIS and overall antisaccade errors (r -.10, p = .65).

Discussion

In the final set of analyses, we explored the relationship between antisaccade performance and schizotypy and the relationship between antisaccade performance and trait impulsivity. Participants with higher scores on the impulsive non-conformity scale of the Oxford-Liverpool Inventory of Feelings and Experiences (OLIFE) inventory made more antisaccade errors than those with lower scores on this scale. There were no differences in correct antisaccade latencies. Participants who scored higher on the Barratt Impulsivity Scale (BIS) had longer correct latencies than those who had a low Impulsivity score. There were no differences in error rate.

These findings support past research where differences in antisaccade performance were found between high vs. low scoring schizotype groups (Gooding 1999; Holahan & O'Driscoll 2005; O'Driscoll et al., 1998) and research that has found a correlation between a schizotypy questionnaire and antisaccade performance (Ettinger et al., 2005). This deficit in high Schizotypes can be likened to the antisaccade deficits found in Schizophrenia patients, (e.g. Fukushima et al., 1998; Hutton et al., 1998). The finding can also be regarded as an inability to maintain the antisaccade 'task set', something that is suggested to be critical to antisaccade performance (Hutton & Ettinger, 2006; Nieuwenhuis, et al., 2004; Reuter & Kathmann, 2004; Roberts et al., 1994).

In keeping with the results of other studies, schizotypy did not impact on correct antisaccade latencies. Previous research has also reported no differences in correct latencies between high and low Schizotypes (e.g. Holahan et al., 2005; O'Driscoll et al., 1998). Interestingly, antisaccade deficits in questionnaire identified Schizotypes as measured with other negative symptom scales (Social Anhedonia, Physical Anhedonia),

have also only showed significant deficits for errors not latencies (Gooding 1999; Holahan & O' Driscoll 1995). It is not immediately clear why there was no relationship between schizotypy scores and correct antisaccade latencies.

We found a small relationship between impulsivity (as measured by BIS) and antisaccade performance, but for correct latencies, not errors. This finding supports past work that has found increased correct antisaccade latencies in patients who normally show a high level of trait impulsivity, such as patients with attention deficit hyperactivity disorder (ADHD) (Mostofsky et al., 2001; Munoz, Armstrong, Hampton, & Moore 2003). Spinella (2004) found that increased antisaccade errors were associated with increased impulsivity scores; however, this relationship was strongest for the attentional impulsiveness sub scale of the BIS. It is possible that we failed to observe a correlation between antisaccade errors and impulsivity because we looked at the overall BIS score rather than the attentional impulsiveness subscale of the BIS.

The finding that OLIFE scores (impulsive non-conformity scale) only accounted for 4% of the variance in antisaccade error rate supports previous research (Smyrnis et al., 2003). However, BIS scores accounted for 21% of the variance in correct latencies, suggesting that impulsivity is an individual difference that is able to explain a large proportion of the variability in antisaccade performance.

General Discussion

Across three sets of analyses we set out to investigate a range of individual differences that would possibly relate to antisaccade performance in healthy participants. The strongest predictor of antisaccade error rate was correct prosaccade latency and the strongest predictor of correct antisaccade latency was trait impulsivity. Differences in antisaccade errors were found between high and low schizotypes. Performance on a go-no go task correlated with antisaccade errors. The operation span task (OSPAN) was the only test of working memory capacity that correlated with antisaccade error rate. Performance on tasks designed to measure speed of processing did not correlate with antisaccade performance.

The results of this paper suggest that individual differences in working memory and personality do not account for a significant proportion of the large variance in antisaccade performance typically observed in healthy participants. Although we did

observe a significant correlation between working memory capacity, as measured by the OSPAN task, and antisaccade error rate, we failed to find a relationship between the OSPAN task and correct antisaccade latencies, contrary to past work (Unsworth et al., 2004). Again, the comparative power of the Unsworth study compared to ours may explain this discrepancy.

As the OSPAN task is generally associated with verbal working memory, we had predicted that working memory tasks that contained a spatial element might prove to be stronger predictors of antisaccade performance. However, we failed to find a relationship between spatial working memory (as measured by the matrix span and symmetry span tasks) and antisaccade performance. These results were somewhat surprising, because an antisaccade trial involves some degree of spatial processing, as the exact co-ordinates of the goal location (opposite to target) must be plotted in a spatial map within working memory. It is possible that in the present study, we simply did not have enough participants, and lacked statistical power.

The finding that performance on the go-no go task correlates with antisaccade performance is important, as it confirms the tasks share similar properties, i.e. response inhibition. When making an antisaccade, or performing a go-no go trial, a degree of inhibition needs to be exerted to not saccade to the suddenly appearing target (antisaccade task) or to avoid responding to 'go' (go-no go task). Previous research has also found a relationship between go-no go performance and antisaccade performance in healthy participants (Spinella, 2002). Our study replicates and extends the results of Spinella (2002), as we have used more go-no go trials and antisaccade trials. One explanation as to why a relationship occurred is because both tasks are sensitive to prefrontal cortex functioning (Spinella, 2002). Taken together, these findings suggest that response inhibition, not spatial working memory, may be more important when exploring potential relationships between working memory and antisaccade performance.

In the second analysis, we explored the relationship between 'speed of processing' and antisaccade performance. Specifically, correct prosaccade latencies correlated with antisaccade errors, that is, those who are faster to make a prosaccade are those who are more susceptible to making an antisaccade error, a result that has been reported previously (Roberts et al., 1994). This finding is critical to current 'competitive race' models of antisaccade performance which argue that the probability of an error is a function of the speed with which activity in correct and error pathways reach the

threshold for a saccade to be triggered (Massen, 2004; Munoz & Everling, 2004). The relationship between prosaccade latencies and antisaccade errors was supported by the fact that prosaccade latencies accounted for 36% of the variance in antisaccade errors, suggesting that prosaccade performance is critical to understanding antisaccade performance. The correlation may have been stronger than in previous work (Roberts et al., 1994), because we used a mixed pro/antisaccade design, whereby activity in the prosaccade pathway would have remained comparatively high throughout the task, as the status of each trial (prosaccade or antisaccade) was not known until the cue indicated this.

We found no relationship between antisaccade performance and a 'motor speed' task (finger tapping task) nor between antisaccade performance and 'internal processing speed' tasks, (letter comparison task & digit symbol substitution task). It is difficult to know exactly why the speed of processing tasks used here did not relate to antisaccade performance. As mentioned in earlier discussions, small sample sizes may have contributed to the lack of relationship between 'speed of processing' and antisaccade performance.

In the third analyses we investigated the relationship between antisaccade performance and personality, using questionnaire identified schizotypal participants and impulsive participants. Although overall schizotypy scores did not correlate with antisaccade performance, the impulsive non-conformity subscale (from the Oxford-Liverpool inventory of feelings and experiences) revealed that participants with a high score made more antisaccade errors than those with a low score. This finding supports past work that has also found antisaccade deficits in questionnaire identified Schizotypes (Ettinger et al., 2005; Gooding 1999; Holahan & O'Driscoll, 2005; O'Driscoll, et al., 1998). It is believed that this decline in performance in high schizotypes compared to low schizotypes, or healthy controls, is because of a deficiency in working memory ability and this has also been found in high schizotypes on tests of spatial working memory (Park & McTigue, 1997). The working memory deficiency in schizotypes can be equated to working memory deficits shown by schizophrenic patients (Hutton et al., 1998; Reuter & Kathmann, 2004) and past research has implied that deficits in working memory can mediate antisaccade performance (Hutton et al., 2004). Our findings support this view and suggest that working memory deficiencies shown by Schizotypes reflects an inability to activate or maintain the antisaccade 'task set' required for antisaccade performance (Hutton 2008; Nieuwenhuis., et al., 2004).

The finding that schizotypy did not impact on correct antisaccade latencies supports past research (Gooding, 1999; Holahan & O'Driscoll, 2005; O'Driscoll et al., 1998). This may simply suggest that antisaccade errors are more sensitive to schizotypy scores than correct latencies are. Deficits in antisaccade performance are believed to act as a potential marker to developing Schizophrenia (see Levy et al., 1994 for review). Likewise, questionnaire identified schizotypes who show increased antisaccade errors, may be more vulnerable to developing psychosis (Gooding 1999). To our knowledge, our study is the first to find differences in antisaccade errors between high and low schizotypes using an Impulsive non-conformity scale. This dimension of schizotypy is concerned with psychoticism, an important feature of the disorder. It is our view that this method of identifying schizotypy is validated and our findings suggest that the psychoticism dimension of the OLIFE is useful for finding participants who may be susceptible to developing Psychosis.

We found a relationship between Impulsivity (as measured by the Barratt Impulsivity Scale) and antisaccade performance. Specifically participants with a high impulsivity score were slower to make an antisaccade than participants with a low impulsivity score. This supports past research where an association between Impulsivity and antisaccade errors has also been found (Spinella, 2004). Other studies have found increased antisaccade latencies in patients who demonstrate high levels of impulsivity (Mostofsky et al., 2001; Munoz et al., 2003). The apparent relationship between Impulsivity and antisaccade performance may be explained by the increased sensitivity to prefrontal functioning found in both impulsivity (Fallgatter & Hermann, 2001) and the antisaccade task (O'Driscoll et al., 1995). Alternatively, increased correct antisaccade latencies in high-Impulsives may reflect a deficiency in additional processing resources used for the inhibition of an erroneous prosaccade.

In conclusion, we have shown that working memory and personality have modest relationships with antisaccade performance in healthy participants. Having used a range of working memory capacity, speed of processing and personality measures, the strongest predictor of antisaccade error rate was actually correct prosaccade latencies. In other words, participants are more likely to make an antisaccade error if they are fast to make a prosaccade. This relationship is important to current race models of antisaccade performance (Massen, 2004; Reuter & Kathmann, 2004) and suggests that researchers should take into account the underlying processes of prosaccade performance when trying to understand the processes underlying antisaccade performance. The fact that

scores on the go-no go task almost correlated with antisaccade errors supports the relevance of response inhibition when generating an accurate antisaccade. The correlation between OSPAN scores and antisaccade errors replicated past work and adds support to its robustness as a reliable measure of working memory capacity and as a good predictor of antisaccade performance. In addition, we have shown that high schizotypy increases antisaccade error rate and high impulsivity increases correct antisaccade latencies. Further research is required to find predictors that are able to explain large proportions of the variability in antisaccade performance.

Part 6 – General Discussion

As described extensively in the introductory chapter, antisaccade error rate varies enormously within the healthy population. A central aim of this thesis was to explore possible determinants of this variability. The studies presented above have outlined a range of top-down factors and individual differences that have impacted on antisaccade performance. A secondary aim of the thesis was to establish the extent to which the effects of various manipulations and individual differences can be explained within current models of antisaccade performance. Importantly, not all antisaccade findings reported here could be accommodated easily within competitive race models, or parallel process models of antisaccade performance. A broader aim of this thesis was to develop our understanding of the cognitive processes underlying antisaccade performance in healthy participants. In the following sections, the key empirical findings are discussed in the context of the main aims of the thesis, and their implications considered.

6.1. Correct prosaccade latency predicts antisaccade error rate

In paper five, the relationship between antisaccade performance and a number of individual differences was explored in a series of correlational analyses. Somewhat disappointingly, individual differences in working memory capacity (as measured in a variety of ways), personality measures (schizotypy / impulsivity) and processing speed did not explain any significant amount of the variability in antisaccade error rate within the samples tested. Possible reasons for these negative findings are discussed in section 6.6. The one individual difference that was found to have a strong relationship with antisaccade error rate was prosaccade latency. That is, those healthy participants who were fastest to make a prosaccade were those more likely to make an antisaccade error. This association was observed in data collected for article 1 (and reported in the published version of this article) but is reported in this thesis in article 5, which considered other individual differences. Correct prosaccade latencies accounted for as much as thirty six per cent of the variability in antisaccade errors, making it by far the strongest predictor of antisaccade performance detected in the experiments reported in this thesis.

The significance of the relationship between correct prosaccade latencies and antisaccade error rate is two-fold. Firstly, the relationship between prosaccade latencies and antisaccade errors supports recent competitive race models of antisaccade performance (Massen, 2004; Munoz & Everling, 2004; Reuter & Kathmann, 2004) and accumulator models of antisaccade performance (Cutsuridis et al., 2007), which predict a close link between speed and accuracy regarding antisaccade performance. Secondly, the correlation suggests that in order to understand variability of antisaccade performance in healthy participants, we must endeavour to understand the individual differences that underlie variability in prosaccade performance too.

As detailed in section 4.4.2 of the introduction, according to parallel processing models (competitive race model) or accumulator models of antisaccade performance, the probability of making an antisaccade error is dependent on the speed of which activity in the separate prosaccade (error) and antisaccade (correct) pathways reaches the threshold for a saccade to be triggered. If the rate of rise in prosaccade activity is faster than the rate of rise in antisaccade activity, then an antisaccade error is likely to occur (erroneous prosaccade to target stimulus). Therefore, the correlation between prosaccade latencies and antisaccade errors we observed is exactly what would be predicted by parallel processing models.

Other papers have also reported a relationship between prosaccade latency and antisaccade error rate in healthy participants (e.g. Ethridge, Brahmbhatt, Gao, McDowell, & Clementz, 2009; Roberts et al., 1994), but not all do (see Harris, Reilly, Keshavan, & Sweeney, 2006). The earliest correlation appears to be in Roberts et al. (1994). Over three experiments, they found that individual differences in the prosaccade latencies were negatively correlated with the proportion of antisaccade errors when antisaccades were performed alone and when antisaccades were performed with an additional task. They suggested that faster prosaccade responding could have been reflected by between-participant differences in the prepotency of the cue on antisaccade trials.

The authors argue that although variability in the inhibition of the pre-potent response exists, this inhibition may occur if the appropriate working memory resources are activated to achieve the task goal (i.e. antisaccade to the opposite location of the impending target). This interpretation fits into goal activation accounts of antisaccade performance (Eenshuistra et al., 2004; Nieuwenhuis et al., 2004; Reuter & Kathmann, 2004), which suggest that differences in the inhibition of the prosaccade is a by-product

of individual differences in the ability to activate and maintain the task goal in working memory. This is an opposing view to the two-stage model of antisaccade performance which argues that the inhibition of the prosaccade is a separate requirement, not a by-product of another action.

The correlation between prosaccade latencies and antisaccade errors found in the present study was stronger than in the study by Roberts et al. (1994). One possible reason for this is that the present study used a mixed pro/antisaccade design. In this type of design, prosaccade activity would have remained comparatively high throughout the task, because the trial status (prosaccade or antisaccade) was not known until the cue indicated this (see article 5, analysis 2). Whereas in single blocks of antisaccades, it could be argued that prosaccade activity would be comparatively lower. Indeed, we failed to observe a significant relationship between prosaccade latencies and antisaccade errors when comparing prosaccade latencies and antisaccade errors from separate blocks (see article 5, analysis 2), although the correlation was in the expected direction. This lack of relationship between prosaccade latencies and antisaccade errors when comparing single blocks supports past work (Harris et al., 2006).

The relationship between prosaccade latencies and antisaccade errors suggests that prosaccade performance needs to be considered when understanding antisaccade performance in healthy participants and this can only be achieved if researchers begin to report these correlations.

6.2. Strategic influences of antisaccade performance

Article 3 provided clear evidence that antisaccade performance can be influenced by top-down strategies. Specifically, healthy participants were able to modulate their antisaccade performance according to different task instructions. The key finding to emerge from this study was that when participants were instructed to delay making an antisaccade, (exp. 1 & 2) or were told to delay making the antisaccade until an auditory cue was given (exp. 3), the number of antisaccade errors they made fell significantly compared to when they were instructed to make antisaccades as quickly and as accurately as possible. This reduction in antisaccade errors was accompanied by an increase in correct antisaccade latencies (exp. 1 & 2). On the face of it, this latter finding is somewhat difficult for current competitive race models of antisaccade

performance to accommodate (Massen, 2004; Munoz & Everling, 2004; Reuter & Kathmann, 2004) as these models predict that a manipulation that increases correct antisaccade latencies should also increase antisaccade error rate. As outlined in section 4.4.3 of the introduction, race model accounts of antisaccade performance argue that a manipulation capable of increasing correct antisaccade latencies should also increase antisaccade errors, providing the manipulation does not affect prosaccade latencies to the same degree. If correct antisaccade latencies are slower, and prosaccade latencies unaffected, then it is more likely on any given trial, that an erroneous prosaccade to the target will be made before the correct antisaccade.

If both prosaccade and antisaccade activity is increased or decreased to a similar degree, then antisaccade error rate should remain unchanged, as neither pathway has gained an advantage from the manipulation. This hypothesis was tested in the second and third experiments of article 3, which included a prosaccade condition, in order to ascertain the effects of task instructions on both pro and antisaccade performance. In experiment 3, participants performed separate blocks of fixation trials, prosaccades, antisaccades and delayed versions of these tasks where the pro or antisaccade had to be made when an auditory cue was heard (see article 3, exp 3 methods). The results of this study were in line with competitive race model predictions. As mentioned above, race model accounts of antisaccade performance suggest that any manipulation which differentially affects prosaccade and antisaccade latencies will in turn impact on antisaccade error rates. This was the case in experiment 3, as prosaccade latencies were significantly increased under ‘delay’ instructions compared to standard instructions, whereas antisaccade latencies were similar under delay and standard instructions. This resulted in a reduction in antisaccade direction errors under delay instructions compared to standard instructions, as predicted by the model.

In experiment 2, participants performed prosaccades and antisaccades under a range of different task instructions, including standard, accuracy, speed and delay (see article 3, exp 2 methods). The results of this study were not in line with race model predictions. The amount of time it took participants to make a prosaccade or an antisaccade under delay instructions was longer than the time it took for them to make either saccade under standard instructions, yet antisaccade errors were reduced in the delay condition. In other words, prosaccade and antisaccade latencies were affected to a similar degree; therefore antisaccade errors should have remained unchanged, as neither pathway has gained an advantage from the manipulation.

One possible explanation of this finding draws on the work of Reuter, Jager, Bottlender, & Kathmann (2007). These authors also found that antisaccade errors were reduced in a delayed prosaccade task. They suggested that in situations where fixation is required (e.g. under the delay instructions used in the present study), the competition arises between fixation neurons and prosaccade neurons (as opposed to between neurons supporting the prosaccade and those supporting the antisaccade). They argued that errors are more likely to be reduced in this situation because fixation provides a strong source of competition for the erroneous prosaccade; meaning that activity in the prosaccade pathway is less likely to reach the threshold for triggering a saccade. Therefore the finding from experiment 2 that increased antisaccade latency was associated with reduced errors can be accommodated by race model accounts of antisaccade performance, if these models are extended to allow the race to be between activity in fixation neurons and neurons responsible for the competing erroneous prosaccade.

The use of top-down strategies is also relevant to goal activation accounts of antisaccade performance. As discussed in section 4.4.4, goal activation accounts of antisaccade performance suggest that antisaccade error rates will reflect the extent to which the relevant task goal can be activated within working memory. Within this framework, it could be argued that antisaccade errors were reduced in 'delay' conditions in paper 3 because delay instructions change the nature of the task goal. In standard antisaccade trials, at target onset, the goal is to 'make an antisaccade' as quickly and as accurately as possible, and this goal competes with the pre-potent response to make an erroneous prosaccade to the target. In delay conditions, however, the goal at target onset now becomes 'maintaining fixation'. The goal to maintain fixation at target onset is a better competitor to the erroneous prosaccade, because active fixation neurons will inhibit saccade neurons in the superior colliculus (Reuter et al., 2007; Taylor & Hutton, 2009). The following section considers other evidence from the experiments reported within this thesis that supports goal activation accounts.

6.3. Task instructions & working memory load interact with antisaccade error awareness

The fourth article of this thesis found evidence that manipulations known to affect antisaccade error rate (task instructions and working memory load) also influence antisaccade error awareness. In the first experiment, instructions to delay making an antisaccade reduced the proportion of aware errors, compared to when participants received 'standard' and 'speed' instructions. In the second experiment, performing antisaccades with an additional task (spatial tapping, or random number generation task) also increased aware errors compared to a condition where only antisaccades were performed. In both studies, the proportion of errors that participants were 'unaware' of remained unaffected by the manipulations.

In support of previous research, we also found a number of differences between aware and unaware antisaccade errors, notably reduced amplitudes and faster correction times in unaware compared to aware errors. Previous studies have found evidence that aware and unaware antisaccade errors are also dissociable by their neural signatures (Endrass et al., 2007; Nieuwenhuis et al., 2004). Taken together, these differences suggest that the relationship between goal activation and unaware errors is different to that of the relationship between goal activation and antisaccade errors. Many researchers believe that antisaccade errors are a consequence of a failure to activate the task goal, or 'antisaccade set' (intention to make antisaccade) within working memory and this intention can be considered as a top-down requirement (e.g. Eenshuistra et al., 2004; Nieuwenhuis et al., 2004). Based on this, manipulations capable of altering the level of top-down control needed for antisaccade performance should impact on antisaccade error rates. It is therefore possible that unaware antisaccade errors were unaffected by the top-down manipulations used in article 4 because unaware errors do not necessarily arise from a failure to activate the task goal.

The difference in the time it took to correct aware and unaware antisaccade errors in this article may reflect a difference in the speed of activity in the separate prosaccade and antisaccade pathways. As unaware errors were corrected in a shorter amount of time compared to aware errors, it is likely that on unaware trials, prosaccade activity (exogenous signal) only just reached the threshold for saccade triggering before antisaccade activity (endogenous signal) resulting in an erroneous prosaccade to the

target stimulus followed closely by a correct antisaccade to the opposite location of the target. Whereas on aware trials, prosaccade activity reached the threshold for saccade triggering well before antisaccade activity got there. These results further support the idea that exogenous and endogenous neural signals can be programmed in parallel (Godjin & Theeuwes, 2002; Massen, 2004; Reuter & Kathmann, 2004; Theeuwes, et al., 1998).

6.4. Incentives have modest effects on antisaccade performance

In article 2 we explored the role of motivation in antisaccade performance by giving participants various incentives. Relatively modest effects of incentive on antisaccade performance were observed across three experiments. However, there were also some inconsistencies between studies, and overall the results did not help to resolve the inconsistencies observed in other studies exploring the effect of incentives on antisaccade performance (e.g. Blaukopf & Di Girolamo, 2006; Hardin et al., 2007; Jazbec et al., 2006).

One important novel finding that did emerge was that there was a trend for non-financial incentives (i.e. simple verbal feedback on trial performance) to reduce antisaccade errors compared to receiving no incentive (exp. 1a & 1b) although the reduction in errors was only significant when feedback was given with monetary reward in the same condition (exp. 2).This finding suggests that feedback, not monetary reward is better at improving participants' motivation, by increasing goal activation and reducing antisaccade errors, compared to receiving no incentive. The finding that monetary reward alone reduced correct latencies not antisaccade errors (exp. 1a, 1b & 2) is surprising. Presumably, a high rewarding incentive would encourage activation and maintenance of the task goal, resulting in a decrease in errors. However, the present results suggest that participants did not perceive monetary reward as something worthwhile enough to motivate performance, and maintain the task goal sufficiently to reduce errors.

The fact that trial by trial audio/visual feedback modulated antisaccade performance in article 2, could suggest that participants were using strategies to alter their performance based on the feedback they were given. Models of cognitive control (Botvinick, Braver, Barch, Carter, & Cohen, 2001; Dehaene et al., 2003; Miller &

Cohen, 2001) argue that participants do adjust current performance on the basis of the outcome of the previous trial. One such strategy is 'post error slowing' (Botvinick et al., 2001; Hodgson Golding, Molyva, Rosenthal, & Kennard, 2004; Rabbitt, 1966) in which participants demonstrate increased correct latencies on trials following an error. A recent trial by trial analysis study found post error slowing on antisaccade trials when the previous error trial was corrected slowly, but not quickly (Tatler & Hutton, 2007). In this study though, participants were not given any feedback on their performance. It would be interesting to conduct a trial by trial analysis of antisaccade performance when trial by trial feedback on error rate is given. Firstly, this could confirm that trial by trial feedback is important to improving antisaccade performance because participants use information on previous trials to monitor ongoing behaviour on current trials and secondly, confirm whether participants only use post error slowing when the previous antisaccade error was corrected slowly. It is clear from article 2 that feedback motivates participants to use task information to modulate antisaccade performance.

6.5. Correlates of antisaccade performance

One possible source of variability in antisaccade error rate are individual differences in factors such as working memory capacity, processing speed and personality traits such as schizotypy and impulsivity. In article 5, a series of correlational analyses was conducted on data collected in experiments 1-4. As discussed earlier in the second analysis of article 5, a key finding to emerge was that prosaccade latencies correlated with antisaccade errors. Surprisingly, given previous research (described in section 4.4.8.) only modest relationships were found between antisaccade performance and a measure of working memory capacity (operation span task) and antisaccade performance and measures of personality (schizotypy, impulsivity). Interestingly, no relationship was found between antisaccade performance and measures of processing speed.

Although modest, the correlation between antisaccade errors and scores on the operation span task (OSPAN) (higher OSPAN scores associated with fewer errors) provides some support for the hypothesis that working memory processes are involved in correct antisaccade performance. As mentioned in earlier discussions (see article 5), it is likely that a smaller correlation was observed in this study because other studies used

a screening procedure to identify participants in the upper and lower quartiles of the OSPAN distribution (Unsworth et al., 2004), whereas in the present thesis, the experiment used an opportunity sample, comprising mainly psychology undergraduates, and as such, the range of scores may have been comparatively narrow.

The trend towards a correlation between antisaccade error rate and schizotypy scores found in article 5 supports numerous studies which have also found this relationship (e.g. Ettinger et al., 2005; Gooding, 1999; Holahan & O'Driscoll, 2005; Larrison et al., 2000; O' Driscoll et al., 1998). With the exception of Larrison et al. (2000), most of these studies have observed a relationship between schizotypy scores and antisaccade errors on 'step' trials, not 'gap' trials. Therefore it was not surprising that the correlation found in the present study only approached significance, as gap trials were used in this study. Another important point to note is that previous studies have only found associations with antisaccade error rate and scales designed to tap positive or negative symptoms of schizotypy. To our knowledge, we were the first study to find a relationship between antisaccade error rate and a scale designed to tap asocial aspects of schizotypy (impulsive-non conformity scale). This finding further strengthens antisaccade error rate as a useful behavioural marker for the onset of schizophrenia.

It is plausible that the similarities in antisaccade deficits shown by schizophrenic patients (see Hutton & Ettinger, 2006) and participants who demonstrate elevated levels of schizotypal traits (such as in article 5), are due to a working memory deficiency in both populations. Increased antisaccade errors in high scoring schizotypes may reflect an inability to sufficiently maintain the intention to make an antisaccade within working memory. This deficiency in working memory is supported by several studies which have found performance deficits on a range of other working memory tasks in schizotypes (e.g. Kopp, 2007). Although schizotypy scores were only accountable for 4% of the variance in antisaccade error rate, this study has at least attempted to identify an individual difference within healthy participants that might explain this variance.

In addition to the relationship found between antisaccade error rate and schizotypy, a correlation between correct antisaccade latencies and elevated levels of impulsivity was also found inarticle 5. That is, high impulsivity scores were associated with increased latencies. Again, the reason for this impairment in antisaccade performance may be due to a working memory deficit, akin to the working memory deficits shown in high schizotypes. This is supported by the fact that many studies have found working memory deficits in high impulsives on a range of cognitive tasks (e.g.

Keilp et al., 2005). As discussed earlier in the discussion of paper 5, these working memory deficits may reflect a deficiency in additional processing resources used for the inhibition of an erroneous prosaccade. In addition, other studies have observed relationships between impulsivity and other tasks which measure 'response inhibition', such as the go-no go task (e.g. Horn, Dolan, Elliott, Deakin, & Woodruff, 2003). This suggests that impaired antisaccade performance is associated with impulsivity because high impulsives demonstrate impaired response inhibition, consequently resulting in slower antisaccade latencies compared to low impulsives. Interestingly, studies have found that prosaccade errors were similar for high impulsives compared to low impulsives (Carr et al., 2006; O'Driscoll et al., 2005) further suggesting that differences in task performance between high and low impulsives are more likely to occur if 'response inhibition' is required. Finally, increased sensitivity to prefrontal functioning found in both antisaccade performance and impulsivity (Fallgatter & Hermann, 2001; O'Driscoll et al., 1995) confirms this relationship.

6.6. Theoretical Implications

It is important to consider how the findings from the present thesis relate to the predictions of current models of antisaccade performance. As mentioned earlier (see section 4.4.2) parallel programming or 'competitive race' models suggest that the ability to make a correct antisaccade is dependent on whether there is sufficient activation in the neural pathway supporting the antisaccade program. A sufficient level of antisaccade activation will occur if activity in this pathway reaches the desired threshold for triggering a saccade before activation in the competing prosaccade program. A prediction of this model is that a manipulation capable of differentially affecting prosaccade activation and antisaccade activation (e.g. increasing prosaccade latencies and reducing antisaccade latencies) should then impact on the probability of an antisaccade error being made, as the manipulation will influence the likelihood of either the prosaccade or antisaccade program reaching the threshold for saccade triggering first. Conversley, a manipulation which influences prosaccade and antisaccade activation to a similar degree should have no impact on the probability of an antisaccade error being made.

The results from article one are not easily accommodated by predictions of race model accounts of antisaccade performance. For example in both experiments, compared to uncued trials, cueing the location opposite to the target stimulus for prosaccades and antisaccades resulted in slower latencies for prosaccades and faster latencies and increased errors for antisaccades. To some extent this finding supports race model predictions, as prosaccade and antisaccade latencies have been differentially affected and this has impacted on the proportion of antisaccade errors made. However, the race model would predict that faster antisaccade latencies should lead to reduced errors, as activity in the antisaccade pathway has been speeded up encouraging the antisaccade program to reach the desired threshold for triggering a saccade before the competing proaccade program.

Another finding from the first paper which is difficult for race models to accommodate is the fact that longer instruction lead time (ILT) led to reduced prosaccade and antisaccade latencies and errors. Again to some extent this finding supports race model predictions, as both latencies and errors were reduced for antisaccades. However, as prosaccade latencies and antisaccade latencies were affected to a similar degree, there should have been no change in antisaccade errors. It is not entirely clear why these findings do not support race model predictions, but it is possible to explain these error rate findings in terms of goal activation accounts of antisaccade performance.

As discussed earlier (see section 4.4.4) goal activation accounts of antisaccade performance emphasise the importance of activating the task goal in working memory, which has clear parallels to adopting an ‘antisaccade task set’ in order to perform successful antisaccades. These accounts suggest that if task relevant information (e.g. the intention to make antisaccade) is adequately maintained within working memory, then this should reduce the likelihood of an erroneous prosaccade to the target being made. It is possible that antisaccade errors were increased when trials were cued differently to the target because this cue made it more difficult to sufficiently maintain the task goal (antisaccade to the location opposite to the target) within working memory. It may therefore be more difficult to maintain the task goal when this type of cue is used, as it becomes a form of ‘task irrelevant’ information which damages the processes needed to make an accurate antisaccade. The relationship between instruction lead time and antisaccade performance in the first article suggests quite simply that

activation and maintenance of the task goal is better when you have more time to prepare.

The results from article 2 suggest some inconsistencies in terms of supporting race model predictions of antisaccade performance. On the one hand, compared to when participants received no incentive, antisaccade latencies were faster and antisaccade errors reduced (all be it not significantly) when a financial incentive was given (exp 1a) and when feedback was given (exp 1b), which is in line with race model predictions. On the other hand, several findings from the 2nd article failed to support the predictions of the race model. For example in experiment 1b, a group of participants who were given a financial incentive had faster antisaccade latencies compared to a group of participants who were given no incentive. Critically, error rates were not reduced in the financial incentive group which contradicts the race model prediction, as a reduction in antisaccade latencies should lead to a reduction in antisaccade errors. It is however important to note that race model accounts of antisaccade performance make strong predictions concerning the relationship of prosaccade performance and antisaccade performance. In order for these predictions to be sufficiently tested, prosaccade performance should also be measured. Therefore it is difficult to say if the results of article 2 can or cannot be accommodated by parallel programming models of antisaccade performance.

It was assumed that goal activation would be increased when a financial incentive was given to participants in article 2, but it is not entirely clear why financial incentives were unable to significantly reduce antisaccade error rates in all three experiments. The fact that errors were only significantly reduced when audio/visual trial by trial feedback and a financial incentive were given together (exp 2), suggests that participants' level of motivation was enough to appropriately activate and maintain the task goal. The amount of financial incentive given in the present study may have simply been too low in terms of increasing motivation and then enhancing goal activation in antisaccade performance. Another reason why goal activation may not have been improved by financial incentives alone is because the expectancy of actually receiving the financial incentive may have been low. Several participants reported that they were unsure as to whether they would actually receive any financial incentive they won during the tasks, and this uncertainty would have lowered reward expectancy, which in turn could have reduced the motivation needed to maintain goal activation.

The results from article 3 are difficult for competitive race model accounts of antisaccade performance to accommodate. In experiment 1, despite an increase in antisaccade latencies under 'accuracy' instructions, antisaccade errors remained unchanged compared to standard instructions. Similarly, faster latencies were made under 'speed' instructions, but with no change in error rate. These findings suggest that the relationship between speed (latencies) and accuracy (errors) in antisaccade performance is not as closely linked as proposed by race model accounts. However, under 'delay' instructions, antisaccade latencies and errors were both altered (all be it not in the predicted direction), suggesting a relationship does exist between antisaccade latencies and errors, but only in certain conditions. Although these findings appear to have contradicted race model predictions though, the lack of prosaccade data makes it difficult to be certain that this was the case.

In the following experiments of article 3, prosaccade performance was measured but the results still contradict race model predictions. The antisaccade findings from experiment 2 were consistent with the antisaccade findings from the first experiment, in that antisaccade latencies and errors were both altered under 'delay' instructions. This should not have been the case as delay instructions increased both prosaccade and antisaccade latencies, thus neither activity supporting the error proaccade nor activity supporting a correct antisaccade had any advantage in the race to reach the threshold for triggering a saccade. The results from experiment 3 showed that despite differences in prosaccade and antisaccade latencies under delay instructions, antisaccade errors were either unchanged (jump the gun errors) or reduced (direction errors), which does not support the predicted direction of results outlined by the race model. An explanation of these results in terms of a modified version of the race model will be addressed later in this section.

The finding that delay instructions reduced antisaccade error rate compared to standard instructions is important as it suggests that participants' goal activation is greater when instructed to delay making an antisaccade. This finding is comparable to the effect of instruction lead time on the proportion of antisaccade errors made in article 1. As mentioned earlier, participants made fewer antisaccade errors when they were given more time to prepare the antisaccade as presumably a longer amount of preparation time allowed them to be better at activating the task goal within working memory. It is therefore possible that participants were also able to appropriately activate and maintain the task goal under delay instructions, as the requirement to delay meant

that antisaccades in this condition were slowed and therefore more controlled in terms of reducing the possibility of making an erroneous prosaccade to the target. The period of delaying the antisaccade gave participants a good opportunity to consistently access the task goal. The finding that delay instructions reduces antisaccade errors compared to standard instructions suggests that goal activation on antisaccade performance can be enhanced under certain conditions.

Finally, and consistent to previous articles in this thesis, the results from article 4 do not appear to conclusively support predictions of the race model. In the first experiment of this article, although a relationship was found between latencies and errors, it was not in the predicted direction (latencies reduced and errors increased under speed instructions, latencies increased and errors reduced under delay instructions). In the second experiment, the relationship between latencies and errors was in the expected direction, as performing antisaccades with a spatial tapping task led to slower latencies and increased errors compared to when antisaccades were performed alone. A similar result was found when participants performed antisaccades with a random number generation task, although the increase in latencies was not significant. These findings confirm that race model accounts are not able to explain all findings of antisaccade performance, although in line with article 2 of this thesis, no prosaccade data was measured here, making it difficult to know if the data does or does not fit in with race model predictions.

The finding that antisaccade errors were increased when participants simultaneously performed antisaccades with an additional task suggests that goal activation is jeopardised under these circumstances. The tasks used in this experiment (spatial tapping and random number generation task) require working memory processes which are also associated with antisaccade performance. Thus accessing the task goal (make an antisaccade to the opposite location of the suddenly appearing target) is difficult to do because in dual-task situations there is an increased level of task irrelevant information (e.g. spatially tapping) which has to be controlled.

Up to this point the current models of antisaccade performance have been discussed as separate entities. However, it is important to consider how they overlap in terms of explaining antisaccade findings in healthy populations and how race model accounts could be adapted to explain antisaccade performance in healthy participants. In terms of both the race model and goal activation accounts, it could be assumed that better goal activation will lead to an increased level of baseline activation and better

goal maintenance will lead to a greater increase in the rate of rise of activity supporting the antisaccade pathway. However, it is difficult to know if antisaccade findings which support race model predictions are occurring solely because of increased baseline activation, or because of a lowered saccade triggering threshold, or a faster increase in the rate of rise in activity. It is also plausible that a combination of all of these things occurs.

Whatever the relationship between neural activation and antisaccade performance, the findings of the present thesis imply that the predictions of the race model are not suitable for explaining antisaccade findings in all conditions. Therefore, the race model should be modified to account for additional antisaccade findings. As discussed earlier in the 3rd article of this thesis, a modified version of the race model would be necessary for explaining antisaccade performance under 'delay' instructions. Drawing on the ideas of Reuter et al. (2007), we believe that during a delayed antisaccade trial a race does exist between competing pathways, but this competition may not necessarily be directly between activity in the prosaccade pathway and activity in the antisaccade pathway. In fact the competition may exist between fixation activity and prosaccade activity, as instructions to delay making an antisaccade encourage greater levels of fixation activity whereby participants fixate for longer at the central point of the screen compared to standard instructions, which do not encourage fixation. Antisaccade errors are reduced in such situations as fixation provides a more efficient source of competition for the erroneous prosaccade, reducing baseline activity sufficiently that the likelihood of prosaccade activity surpassing threshold after target onset is reduced. A more appropriate competitive race model will be one which recognises that competition can exist between neural systems supporting fixation and those supporting the incorrect response, as well as competition between neural systems supporting the incorrect prosaccade and correct antisaccade.

6.7. Limitations & Future Research

With the benefit of hindsight, and the experience gained over the course of the PhD, there are a number of limitations in the experiments that future research should address. For example, in the first series of experiments (which included exp 1a from article 2), it is possible that no effects of incentive on antisaccade errors were found due

to a lack of experimenter experience. Specifically, a lack of appreciation for the importance of consistency in instructions, and the importance of strategic influences (as revealed by the results of paper 3) may have resulted in participants approaching the task with varying levels of motivation, and differing strategies for successful task performance. In later experiments, experimental procedures will have been more consistent, partly due to experimenter experience.

Another limitation is the comparatively small sample sizes used for the correlations in article 5. With the exception of the correlation between antisaccade errors and OSPAN scores ($N = 58$), all other correlations were analysed using smaller samples ($Ns < 36$). This may have limited the chance of observing significant correlations. In addition, these small sample sizes make any significant correlation found in the present research questionable, e.g. between correct antisaccade latencies and impulsivity scores. The fact that the correlation between prosaccade latencies and antisaccade errors was only significant in the larger sample of participants (see article 5, analysis 2), supports the possibility that larger sample sizes would have increased the chances of observing more significant correlations between antisaccade metrics and the various measures used in article 5. Future research should aim to conduct a large scale correlational analysis between antisaccade metrics and an extensive range of working memory measures. As well as error rate and latencies, these metrics could include spatial accuracy, as this may provide important information concerning the functioning of the neural systems supporting spatial working memory.

An inconsistency in the present thesis was that the two reported correlations between prosaccade latencies and antisaccade errors in article 5 had differing results. The first reported correlation (taken from article 1, exp. 1) showed a significant relationship between prosaccade latencies and antisaccade errors, but the second reported correlation (article 3, exp. 2) did not. As mentioned earlier, it is likely that this difference occurred because the two data sets we used had different designs (prosaccades and antisaccades were performed in mixed blocks in the significant sample and separate blocks in the non significant sample) and as a result prosaccade activity was more relevant to antisaccade activity in the significantly reported correlation.

A further limitation of this thesis was that prosaccade data was only recorded in four of the ten experiments presented here (article 1, exp. 1 & 2; article 3, exp. 2 & 3). Although an attempt was made to interpret the findings reported in this thesis in light of predictions of competitive race model accounts of antisaccade performance, it is

difficult to do this extensively without prosaccade data, given that these models make strong claims about the outcome of antisaccade error rate based on the effects of manipulations on both pro and antisaccade latencies. It would have been beneficial to have included a prosaccade task in all experiments, in order to comprehensively test race model predictions. For example, in article 2 (exp. 1b) an inconsistent pattern of results was found, as participants who received a financial incentive, were significantly faster to make a correct antisaccade but made a similar amount of antisaccade errors than those who received no incentive. Race model accounts of antisaccade performance would predict that a reduction in antisaccade latencies should lead to a reduction in errors (see section 4.4.2). However, even if latencies and errors had been reduced in the financial incentive condition, this finding cannot be accommodated by the race model prediction without first measuring the effect of the incentive on prosaccade performance. If the incentive influences prosaccade latencies differently to antisaccade latencies in this condition, then the race model prediction is supported by the results.

Some of the stimulus properties from the first paper of this thesis differed from the stimulus properties of the other papers. With the exception of article 1, typically the target stimulus was displayed on the horizontal axis without a marker (flanker box) at varying spatial degrees from the central point. However in the first article, flanker boxes were used to outline the location of the target stimulus and the opposite location to this. The flanker box may have facilitated attention by acting as a tool for directing antisaccade eye movements towards the goal location and as a result antisaccade performance may have been improved compared to when no flanker boxes were used. The difference in stimulus set-up may have implications for the relationship that was found between prosaccade latencies and antisaccade errors, as the data was taken from article 1 where flanker boxes were used. It is plausible that this correlation may not have existed if flanker boxes were absent from the stimulus set-up as the task would be more difficult. This assertion is supported by the fact that no relationship was found between prosaccade latencies and antisaccade errors in an additional correlational analysis (taken from article 3), where no flanker boxes were used to outline the goal location. Future work should explore the effects of different stimulus set-ups on antisaccade performance as it is important to have a standardised version of the task which can be used to accurately identify ‘at risk’ individuals for the development of psychosis.

A ‘gap’ paradigm was used in all experiments of this thesis. Although this was consistent throughout the thesis, it is well documented that a gap between the offset of

the central fixation point and the onset of the target stimulus will reduce antisaccade latencies compared to when no gap is used (Fischer, Gezeck, & Hartnegg, 2000). It is possible that some of the reported effects found on antisaccade latencies in this thesis may have been assisted by the use of the gap paradigm because the disappearance of the central fixation stimulus acts as a 'quick release' to disengage attention away from this location thus allowing it to be allocated more rapidly to the location opposite to the target when it subsequently appears. It would be beneficial to include antisaccade data using a different paradigm, for example the 'overlap' paradigm, to check that these effects were not dependent on the type of paradigm used.

In the 3rd article of the thesis, participants were given different verbal instructions (standard, accuracy, speed, delay) when performing antisaccades. Even though every effort was made to standardise the instructions, there is still a possibility that participants' subjective interpretation of the instructions may have influenced results. For example, under speed instructions participants were told that spatial accuracy was not important. Although this part of the instruction was included to encourage a fast response, it may have not had the desired effect and instead drawn attention to the fact that there is a spatial accuracy element to performing an antisaccade. Similarly, under accuracy instructions participants were told that speed was not important for these trials. This creates the same problem as there is a chance that telling participants to not worry about the speed of their antisaccade actually draws more attention to it. The results of article 3 suggest that designing task instructions that specify what participants are supposed to, (i.e. speed instructions which emphasise speed and nothing else) is a difficult task and more piloting is needed to test the suitability of a range of different instructions.

In article 5, some of the measures of individual differences had a limited range of scores. This is an important limitation as for example a correlation between antisaccade errors and symmetry span scores from a restrictive range may be different to a correlation between antisaccade errors and symmetry span scores from a wider range. For the correlation to be generalizable there should be a wide range of scores in antisaccade errors and symmetry span scores. One possible reason as to why there were restrictive ranges in some of these measures is because of the scoring method used. For example, scoring for the operation span task (OSPAN) was calculated as the sum of recalled words for all of the sets in which the entire set was recalled in the correct order, in line with previous studies (e.g. Turner & Engle, 1989). However, previous research

has used alternative scoring methods. In a study by Kane et al. (2004) OSPAN performance was calculated by adding together the number of correct items within each set and then converting this into a proportion-correct score and then the mean proportion-correct score was computed over all sets in the task for the participants' task score. The score for each span task reflected the average proportion correct across the different set sizes. The authors found this scoring method produced the most normally distributed scores compared to 3 alternative scoring methods, including the one used in this thesis. It would have been wise to compare different scoring methods for OSPAN performance to be certain that the chosen scoring method was normally distributed.

One key inconsistency of the present thesis was that spatial working memory had differing affects on antisaccade performance. In article 4, antisaccade errors were increased when participants performed antisaccades with a spatial tapping task (ST) compared to when antisaccades were performed alone. In article 5 we failed to replicate this relationship between spatial working memory and antisaccade performance, as scores on measures of spatial working memory (matrix span & symmetry span) did not correlate with antisaccade error rate. One possible reason for this difference is simply because the ST task was simultaneously performed with antisaccades and the matrix span (MSPAN) and symmetry span (SSPAN) tasks were performed separately. Thus the increased complexity of having to perform the ST task would have caused interference to antisaccade performance. As the antisaccade task was performed separately to the MSPAN and SSPAN tasks, there was no opportunity for these tasks to interfere with antisaccade performance. The fact that no relationship was found between SSPAN, MSPAN and antisaccade performance but antisaccade errors were increased when participants performed spatial tapping suggests that the ST task taxes spatial working memory resources more closely linked to antisaccade performance. Although it would be difficult to explore the effects of dual-task performance on antisaccade error rate using either the MSPAN or SSPAN tasks, it would be interesting to see if there are stronger relationships between other spatial working memory tasks and antisaccade performance and if these relationships are of a correlational nature.

In the 4th article, the key findings were that manipulations which increased and decreased antisaccade errors had no effect on the proportion of antisaccade errors which participants were 'unaware' of and that there were quantitative differences between 'aware' and 'unaware' errors. Although these findings suggest that there are different types of antisaccade errors, it would be interesting to investigate the role of specific

cortical areas in the generation of these errors. This would help identify if aware and unaware errors differ in terms of neural circuitry as well as with saccade size and correction times. To do this, transcranial magnetic stimulation (TMS) could be applied to cortical areas already believed to be involved with antisaccade performance, such as the frontal eye fields (FEF) and posterior parietal cortex (PPC). This would develop an understanding of the role of specific cortical areas in determining whether antisaccade errors reach conscious awareness.

As has been discussed extensively throughout this thesis, the range of antisaccade error rates in healthy participants can be very large. The enormous range of performance within healthy participants is particularly problematic for researchers interested in using antisaccade performance to identify 'at risk' individuals e.g. first degree relatives or those in early stage schizophrenia. This is because if large standard deviations exist, then larger mean differences in performance are required in order for comparisons across groups to become statistically significant. As a consequence, many studies may falsely conclude that antisaccade error rates in 'at risk' individuals, is just as low, or just as high compared to healthy controls, decreasing the utility of antisaccade error rate as a behavioural marker for schizophrenia.

A number of studies have documented a link between antisaccade performance and working memory (e.g. Unsworth et al., 2004). However, the term 'working memory' is used by different psychologists and cognitive neuroscientists to signify a wide range of cognitive processes. Models of antisaccade performance that make reference to it (e.g. goal activation accounts) need to ensure that the precise mechanisms involved are well documented. Future research employing measures of goal activation may help clarify the exact role of working memory in antisaccade performance by exploring additional areas of working memory. For instance, the fact that impaired antisaccade performance is believed to reflect executive dysfunction in schizophrenic patients (see Reuter & Kathmann, 2004 for review), highlights the importance of executive control when generating accurate antisaccades. Therefore, research should attempt to identify individual differences in measures of executive control when trying to account for individual differences in antisaccade error rate in healthy participants. In addition, a systematic programme of research is required in order to establish the extent to which a wide range of personality measures mediate antisaccade performance. Specifically, research should build on the preliminary findings in this thesis, which have

given indication of a relationship between trait impulsivity and antisaccade performance.

6.8. Conclusions

This thesis describes 10 experiments and 3 correlational analyses organised into 5 separate research papers, which together provide a number of insights into why antisaccade error rate varies so much in healthy participants. Several key findings emerged. Firstly, the individual difference that was found to have the strongest relationship with antisaccade error rate was correct prosaccade latency. Specifically, those who were fast to make a prosaccade were also those more likely to make an antisaccade error. A second key finding to emerge from this thesis was that simply instructing participants to delay making an antisaccade resulted in reduced antisaccade errors and increased correct antisaccade latencies compared to when participants received standard antisaccade instructions. The third key finding was that manipulations which increased (working memory load) or decreased (delay instructions) antisaccade errors also impacted on the proportion of these errors which participants were 'aware' of, but had no effect on the proportion of errors that participants were 'unaware' of. Finally, antisaccade performance was only modestly affected by financial and non-financial incentives.

The majority of these results support the predictions of competitive race model accounts of antisaccade performance, outlined in section 4.4.2, particularly if these models are extended to allow the competition to be between activity in fixation neurons and neurons responsible for the erroneous prosaccade. Importantly however, in a number of experiments, the relationship between antisaccade latency and error rate was not as clear cut as race models predict. For example in article 2 (exp. 1b & 2), correct antisaccade latencies were reduced and antisaccade errors remained unchanged when a financial incentive was given alone compared to no incentive. Similarly, in article 3 (exp. 1 & 2), compared to standard instructions, antisaccade errors were reduced under delay instructions even though latencies were increased. These results suggest that in some antisaccade tasks at least, speed and accuracy may not necessarily be closely related.

It is clear from the evidence presented in this thesis that a wide range of factors, such as motivation, strategies and individual differences can influence healthy participants' performance on the antisaccade task. Future studies will be wise to continue to explore these and other factors which may influence antisaccade performance, particularly with the aim of understanding and minimizing the large variability in antisaccade error rate within healthy participants. If this is explored, then deficits in antisaccade performance in other populations can be interpreted to a better degree. In addition, understanding this variability in error rate in healthy participants will ultimately increase the chances of identifying individuals who are 'at risk' for the development of psychosis, and affirm the benefits of using antisaccade error rate as an endophenotype of schizophrenia.

Competitive race model accounts of antisaccade performance have been useful in terms of explaining the underlying processes of antisaccade performance in healthy participants and may provide new insights into these processes, if they are extended. This may provide important insights into individual differences in antisaccade performance and why antisaccade performance in healthy participants is better, compared to certain psychiatric populations, e.g. schizophrenic patients.

In conclusion, this thesis has presented evidence to suggest that antisaccade performance in healthy participants is influenced by a range of top-down factors. The correlation found between prosaccade latencies and antisaccade errors suggests that the cognitive underpinnings of prosaccade performance should be considered when attempting to understand the underlying cognitive processes of antisaccade performance. Furthermore, it is important that a systematic investigation is conducted in order to identify additional factors which can account for the enormous variability often found in antisaccade error rate within healthy participants.

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